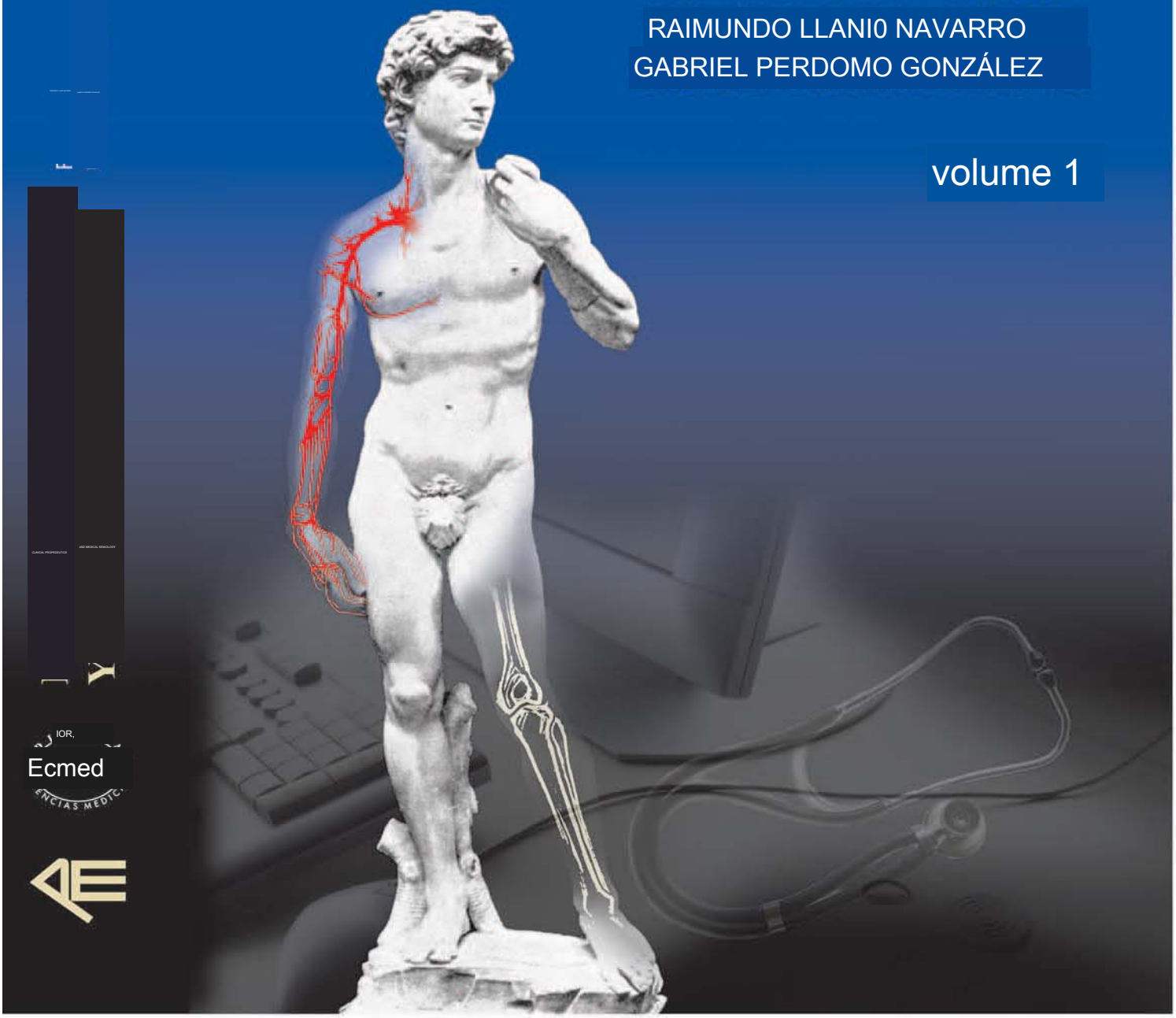
volume 1

CLINICAL PROPEDEUTICS AND MEDICAL SEMIOLOGY

RAIMUNDO LLANI0 NAVARRO
GABRIEL PERDOMO GONZÁLEZ

volume 1



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To my wife Flora Villate, for her help in preparing the book, for her moral support and, above all, for her understanding, patience and sacrifice during thousands of hours dedicated to the completion of this work.

To the students and doctors, past, present and future, owners of the future of medicine, to whom I have dedicated a large part of my life.

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NOTE TO THE READER

In our current medical education system, the student has the opportunity to learn from his first year, in the subject called Introduction to Comprehensive General Medicine, general elements related to the history of medicine, communication, ethics, morality, health, illness, sexuality, family doctors, primary health care and national health programs, in close theoretical-practical connection with the field where he will develop his work, once he graduates.

In the second year of the degree, the student continues, in his first semester, with the preclinical subjects; But already in the second semester of this year, a new subject is incorporated: Introduction to the Clinic, which is taught in primary care and combines the first elements of the clinic, related to the techniques of performing the physical examination in the supposedly healthy adult, with its practice and learning in the place where he will develop his professional activity, once he graduates. On the other hand, it serves as a preliminary step for better and faster learning of the subject Clinical Propedeutics and Medical Semiology, which is taught in the third year of the degree, in secondary care.

But since the technique of the physical examination of an individual without signs of disease is similar to that of a sick adult and for the study of these subjects the book Clinical Propedeutics and Physiopathology has been used, we did not consider it necessary to prepare a separate text for Introduction to the Clinic and rather the publication of a work that separately contemplates the content to be taught in both subjects, structured in a related way, which would help the student's learning, without getting lost in delimiting by itself the border between the normal and the pathological, and at the same time without repetitions in the content.

That is why this work consists of two sections: "Introduction to the Clinic" and "Clinical Propedeutics" itself.

Section I deals with most of the necessary content that allows us to achieve the objective of the Introduction to Clinical subject, which is to achieve the following skills in the student:

Carry out adequate individual communication to undertake the physical examination, without incurring iatrogenesis and taking care of the basic principles of medical ethics.

Perform a complete physical examination on an adult individual without signs of disease, appropriately applying the methodology, basic clinical procedures, and necessary clinical examination techniques.

Correctly record the data collected in each of the parts of the physical examination.

The “Clinical Propedeutics” Section is responsible for the study of the symptoms, signs and syndromes of general, regional and systemic semiology, and for pointing out, without going into depth, the most important paraclinical examinations in the verification of semiological and/or etiological findings: imaging, endoscopies, laboratory tests, biopsies, electrocardiography, etc.; In addition, it complements the physical examination techniques that were partially exposed or not discussed in the previous Section.

We hope that the conceived structure will benefit the reader by facilitating the study of such an important and basic subject in the training of a Medical Sciences professional, that without its complete knowledge it will be impossible to achieve a graduate of optimal quality, competence and performance.

THE AUTHORS

PREFACE

As the years go by, the impact of new discoveries and new technologies occurs, which undoubtedly provide successive generations with different situations than those that existed twenty years before, to cite a comparison.

This means that at the present time we are witnessing a progressive and incessant advance in morphological and functional exploration techniques, in such a way that through various more or less sophisticated and expensive methods we can discover small anatomical alterations and discrete disorders of organic functions that until recently we could not specify.

Although this constitutes an indisputable truth, such advances in diagnostic methods frequently make people forget (especially the young classes of doctors, impressed by technological advances), that classical semiology and, above all, the careful collection and intelligent evaluation of clinical history data, continue to retain their value in modern medicine, and this is also an unquestionable truth.

The Introduction to the Clinic, which deals with the supposedly healthy man and woman, and Clinical Propedeutics, as well as Medical Semiology, form an indivisible part of the student's training at the end of his preclinical stage in the basic sciences and is a vital tool for dealing with the care of sick people, whether in outpatient consultation or in hospitalized patients. We could say that these two subjects constitute the link between basic and clinical sciences; It is therefore its connecting bridge and the basis without which the clinical diagnosis cannot be made.

To better understand the importance of the history and physical examination of the patient, let us point out just three quotes from the great clinicians of the past.

As an anecdote, let us remember E. S. Chweninger's response to Prince Otto von Bismarck, German Chancellor, his patient, who refused to give him the requested information due to lack of time and ordered him to continue his examination without asking him any more questions: Your Excellency should consult with a veterinarian, because he does not ask his patients anything.

The Spanish internist Gregorio Marañón, when referring to the importance of the interTogatorio, stated that: "the device that had made medicine progress the most was the chair."

And Professor Jiménez Díaz, glory of the Spanish clinic, pointed out that: before inspection, palpation, percussion and auscultation (which constitutes the physical examination), the doctor must know how to perform 'listening.'

We invite any doctor or specialist to demonstrate what modern equipment can replace or perform more diagnoses than the chair, through interrogation.

But closely linked to these considerations must be sensitivity, human quality and medical ethics.

Medical acts must always meet two basic conditions: correctness and kindness.

An act is incorrect when it is not technically well performed. If a doctor does not know how to use diagnostic or therapeutic procedures appropriately, we say that he or she uses them "incorrectly." Incorrectness always implies lack of technical sufficiency. That is why a doctor who practices his art incorrectly is called a "bad doctor." There are bad doctors, just as there are bad carpenters, bad car drivers or bad painters. However, you have to know how to differentiate between bad doctors and bad doctors. "Bad doctors don't always identify with bad doctors."

A bad doctor is one who has insufficient or incorrect technical capacity, while a bad doctor is one who uses it poorly because he is morally bad.

A good doctor can be, in turn, a bad doctor, given that sufficient technique does not necessarily imply moral goodness, so both characteristics must be required of the doctor. For this reason, since the times of Roman antiquity, the doctor has been defined as vir bonus medendi peritus, that is, a good man, expert in the art of

cure.

Expertise in the art of healing defines the "technical correctness" of medical practice and turns the person who performs it into a "good doctor"; Human goodness, for its part, defines the moral goodness of the professional and makes him a good doctor. They are two essential factors that demand each other: the lack of one of them is incompatible with the proper exercise of the profession.

Not all human activities demand as much moral elevation from those who practice them as medicine. This is because doctors work with the most precious thing that human beings have, their life and health. Hence the importance that professional ethics has always had in medicine, at least since the origins of the medical tradition in the time of the Hippocratics.

In fact, the ethics of the Hippocratic Oath have not only been the watchword of medical morality for 25 centuries, but also the canon and guide of all other professional ethics.

Professions differ from trades in that in the latter, legal control is sufficient, that is, the subsequent penalization of misdemeanors or crimes. In the medical profession, on the contrary, strict prior control is necessary, a priori, precisely because what is at stake is a value as fundamental as human life. And this prior control can only be ethical. That is why ethics can never be considered by the doctor as something external to his professional activity, but rather as an intrinsic and constitutive element of his.

Therefore: Only a good doctor can be a good doctor.

Years ago, the highest leader of our country, who has always been committed to the health of the population, said that: if in any profession, in addition to great knowledge, great human sensitivity is needed, it is in medicine.

In my opinion, if this is not the case, you cannot be a good doctor because you must never forget that: he represents encouragement and hope for that weak human being who sees his health broken and the least he expects is solidarity and humanism.

I am convinced that sensitivity and tenderness help a patient many times more than with all scientific knowledge.

This summarizes very well the thinking that has guided my life and my performance as a doctor.

Our objective in writing this book has been to provide students and doctors with a tool that is useful and perhaps indispensable in this important field of medicine, which, as Dr. Damodar Peña Pentón, Minister of Public Health, told us, must accompany them "under their arm" during the exercise of their activity.

All doctors talk about the Hippocratic Oath written so many centuries ago, but very few have read or know in detail some of its versions and we believe that it is of great value to point it out here.

Hippocratic Oath

I swear

By Apollo the physician and Asclepius and by Hygieia and Panacea and by all the gods and goddesses, making them judges, may this my oath be fulfilled to the best of my power and discernment. I will esteem the one who taught me this art the same as my parents; He will participate in my maintenance and if he wishes, he will participate in my assets. I will consider their offspring as my brothers, teaching them this art without charging them anything if they wish to learn it.

I will instruct by precept, by speech and in all other ways the sons of him who taught me and the disciples united by oath and stipulation according to the medical law, and not other people.

I will carry out that regime, which according to my power and discernment will be for the benefit of the sick and will separate them from harm and error. I will not give anyone a mortal drug even if it is requested of me, nor will I give advice for that purpose. In the same way I will not give any woman destructive suppositories; I will keep my life and my art away from guilt.

I will not operate (not even by size) on the calculating, leaving the way to those who work in that practice. Whatever I enter into, I will go for the benefit of the sick, abstaining from all willful error and corruption, and from lasciviousness towards women and freemen and slaves.

I will remain silent about everything that in my profession, or outside of it, I hear or see in the lives of men that should not be public, keeping these things in such a way that they cannot be talked about.

Now, if I fulfill this oath and do not break it, may the fruits of life and art be mine, may it always be honored by all men, and may the opposite happen to me if I break it and am perjured.

We find it very useful to end this Preface with a version of the "Advice of Aesculapius" to his son who wanted to be a doctor.

Advice from Aesculapius*

Do you want to be a doctor, my son? This is the aspiration of a generous soul, of a spirit eager for Science. Do you want men to consider you a God who alleviates their evils and drives away their fear? Have you thought well about what your life should be like?

The majority of citizens can, once their task is finished, isolate themselves away from those who are inconvenient; your door will always remain open to everyone; They will come to disturb your sleep, your pleasures, your meditation; You will no longer belong to yourself. The poor, accustomed to suffering, will only call you in case of emergency; but the rich will treat you like a slave in charge of remedying their excesses; Whether because they have indigestion or because they have a cold, they will make you wake up in a hurry as soon as they feel the slightest restlessness; You will have to show interest in the most vulgar details of their existence, decide if they should eat lamb or mutton, if they should walk in this or that way. You will not be able to be absent, nor be sick; You will have to always be ready to come as soon as your master calls you.

Do you have faith in your work to earn a reputation? Keep in mind that they will judge you not by your science, but by the coincidences of fate, by the cut of your cloak, by the appearance of your house, by the number of your servants, by the attention you dedicate to the talks and to the tastes of your clientele. There will be those who will distrust you if you do not come from Asia; others if you believe in the gods; others if you don't believe in them.

Your neighbor, the butcher, the shopkeeper, the shoemaker, will not trust you with his clientele if you are not his customer; The herbalist will not praise you unless you prescribe his herbs. You will have to fight against the superstitions of the ignorant. Do you like simplicity? You will have to adopt the attitude of an augur. Are you active, do you know what time is worth? You will not show annoyance or impatience; You will have to endure stories that start from the beginning of time to explain colic.

* Aesculapius: born in the year 1 BCE. was a Greek physician, native of ancient Bitina in Asia Minor. He was a professor of oratory in Rome (where he is known as Asclepius). but later he graduated as a physicist and dedicated himself to the practice of medicine. He believed that the movement of atoms in the body was the cause of diseases, and he advocated baths, diet, exercise and massage as cures. His numerous followers founded a medical school called Metódica.
After his death, multiple temples were built in his honor with the idea of perpetuating his memory and wisdom.
In the current era it is internationally recognized and is worshiped in the emblem of medicine. As is known, the emblem is represented by an image where you can see a snake (Aesculapius snake) coiled around the rod that serves as a symbol. This European reptile is harmless, brown on the dorsal side and yellow or white on the ventral side. It lives in Spain and part of Europe.

Do you feel passion for the truth? You will no longer be able to tell it. You will have to hide the seriousness of their illness from some, and its insignificance from others, because it would bother them. You will have to hide secrets that you have, consent to seem mocked, ignorant, complicit. You will never be allowed to doubt, under penalty of losing all credit; If you do not affirm that you know the nature of the disease, that you have an infallible remedy to cure it, the masses will go to charlatans who sell the lie they need.

Do not count on gratitude: when a sick person heals, the healing is due to his robustness; If he dies, you are the one who killed him. While he is in danger, he treats you like a God, he begs you, he promises you, he showers you with flattery; As soon as he is convalescing you are already in his way; When it comes to paying for the care you have given them, they get angry and denigrate you.

I pity you if you feel a desire for beauty: you will see the ugliest and most repugnant things in the human species; all your senses will be mistreated. You will have to press your ear against the sweat of dirty breasts, breathe the smell of miserable homes, the very strong perfumes of the courtesans, feel tumors, cure green sores of pus, contemplate the urine, scrutinize the sputum, fix your gaze and your nose on filth, put your finger in many places. They will call you to a man who, bothered by stomach pains, will present you with a nauseating chamber pot, telling you with satisfaction: "thank you I was careful not to throw it away." Remember then that you must seem very interested in that excrement.

Your job will be a Nessus tunic for you: in the street, at banquets, in the theater, in your bed itself, strangers, your friends, your loved ones will talk to you about their illnesses to ask you for a remedy. The world will seem to you like a vast hospital, an assembly of individuals who complain. Your life will pass in the shadow of death, between the pain of bodies and souls, of duels and the hypocrisy that calculates, at the bedside of the dying.

You will see yourself alone in your sadness, alone in your studies, alone in the midst of human selfishness. When, at the cost of many efforts, you have prolonged the existence of some elderly people or deformed children, a war will come that will destroy the healthiest and most robust in the city.
Then, you will be tasked with separating the weak from the strong, to save the weak and send the strong to death.

Think about it while you have time. But yes, indifferent to fortune, to pleasures, to ingratitude; If, knowing that you will find yourself alone among human beasts, you have a soul stoic enough to satisfy yourself with your duty fulfilled without illusions; If you consider yourself sufficiently rewarded with the happiness of a mother, with a face that smiles because it no longer suffers, with the face of a dying man from whom you hide the arrival of death: if you long to know man, to penetrate all the tragedy of his destiny, then become a doctor, my son. *

* Taken from http://fcmjtrigo.sld.cu/doc.htm

I do not want to end without recognizing and thanking all the co-authors, collaborators, Dr. Lorenzo Muñoz Caldas and Dr. Josefa Campanioni Tamayo, First Degree Specialists in Hematology, and those colleagues who in one way or another have been part of the preparation, review and publication of this book, especially Iraida Gumá, Carmen Fleites, María Isabel Otero, Wanda Canals, Professor Baudilio Jardines, the specialized staff of Editorial Pueblo y Educación, and in In general, to all the colleagues who have given us their cooperation, without which this work would not have been able to fulfill its mission.

Prof. RAIMUNDO LLANIO NAVARRO

Havana, 2003

THE PHYSICAL EXAMINATION AS PART OF A CLINICAL RECORD

The clinical file (or medical history) of a person, as the word indicates, is the document where all of the person's data, related to their state of health or illness, is collected in chronological order.

Without going into details that will be discussed in Section II of Propedeutics, the clinical history, regardless of the clinical situation and the place where it is collected (doctor's office, home, outpatient clinic, emergency department or hospital admission room) consists of two primary components: the interrogation and the physical examination.

The purpose of the physical examination is also variable; It can be carried out in response to certain symptoms reported by a patient, either urgently or with sufficient time for a more complete physical examination, or to detect the existence of asymptomatic diseases, as occurs in periodic medical examinations of workers, students or groups at risk.

Due to the variety of places, clinical situations or purposes for which a physical examination is performed, modification of the technique may be necessary, but the general, essential and essential guiding bases of the techniques for performing it on a supposedly healthy individual do not vary in the sick individual, except in the special techniques for diagnosing the signs of illness, which will be discussed in Section II of this work.

Those of us who are used to teaching medicine in a traditional way find it difficult to understand that the physical examination can be taught without having previously learned how to conduct an interrogation. This is because although it is true that for the clinical diagnosis of disease, the interrogation and the physical examination are inextricably linked, and one is not conceived in the absence of the other, when the objective of learning in this first stage is not focused on the clinical diagnosis of the patient, but on the development of learning technical skills that will be completed and consolidated in a second stage, the physical examination can be learned without prior teaching of the techniques of the medical interview and the inter-gatory. We will dedicate the following chapters of this Section to this.

However, we know that in practice there are no defined boundaries between the normal and the pathological, other than from a

didactic; But this is not a negative aspect, quite the opposite. The finding of something that is not normal, while learning the physical examination at this stage, although it should be avoided, does not negate this teaching method. And if the educator stops, does not waste the opportunity, and briefly explains what he has found, it can serve as motivation in the students to search and individually study something that they have seen for the first time and that they will surely never forget.

communication

GENERAL ASPECTS OF THE COMMUNICATION

Communication is the basis of relationships between people and human groups. It can be defined as the process of interaction and exchange of information between men, where some seek to influence the customs, ideas and attitudes of others. That is to say, all communication is, at the same time, a conscious or unconscious intention to modify the other.

For this reason, it decisively influences the quality of medical care and is the most important element in the doctor-patient-family-community relationship.

Communication can be direct (person to person) or indirect (through mass media, such as the written press, radio, television and cinema).

The one who issues the message is considered the " source" (patient), who must prepare and transmit the information, in such a way that the "receiver" (doctor) is able to receive it and "decipher" it; that is, understand it and, in turn, then act as a "source" for the patient.

When this happens it is said that they are in tune."

patient		what you feel		doctor
SOURCE	═══	MESSAGE	═══	RECEIVING
doctor		what advises or determines		patient

Verbal communication

Verbal communication is the form of human communication par excellence. Language is your system of signals, whether oral or

Words, in any language, contain the essential qualities of a phenomenon. For example, the word "sick" (the concept) does not refer to any sick person in particular, but to the qualities that a man must have to be considered as such. This is the meaning of the word. The coincidence of interpreting the meanings in the same terms used by the sender is the basis of good communication.

extraverbal communication

This form of communication is established through extraverbal language (Pavlov's first system of signals) and constitutes the expression or externalization, through motor means, of states and reactions, emotional ones above all.

Gestures, facial expressions, manners and movements in general also have a communicative function. A look can denote joy (or fear), a frown can mean surprise or disapproval; A movement of the head can indicate assent or denial.

COMMUNICATION IN MEDICAL PRACTICE

The interview is one of the two communication techniques most used in medical practice and it has been very correctly said that if it is not used, the medical act has not even begun.

The technique for adequate communication during the individual medical interview could be divided into five basic parts:

- The meeting.
- The interrogation.
- During the physical examination.
- Evaluation of the behavior to follow.
-The farewell.

The technique of the medical interview for the preparation of the clinical history, especially for the interrogation or anamnesis, will be addressed in Chapter 3 of Section II of this

COMMUNICATION IN THE PHYSICAL EXAM

The medical student, who begins his first steps in the clinical area with learning the techniques of the physical examination, has a special communication with the subject, who is at the same time the object of his learning.

Generally, the student will be accompanied by the doctor responsible for the subject's medical care and the student's tutorial teaching; For the moment, he will not take the anamnesis nor is he qualified to evaluate the conduct to be followed, so communication during the interview will take place in three main moments:
the meeting, during the physical examination and at the farewell.

This type of communication differs somewhat from the classic medical interview, from a point of view of form, but not of content, and therefore, the skill learned will also be useful for carrying out the complete medical interview, later as a student and throughout your life as a professional.

The meeting

Most likely, the tutor doctor will introduce the student, starting the meeting. If this does not happen or at some point the student has to approach a patient alone, the meeting should always begin with greeting and introduction.

The greeting should be done by shaking the interlocutor's hand, looking him in the eyes, transmitting the warmth of a smile, and with respect and courtesy in accordance with the age of the subject.

Next, both the student and the doctor who sees a patient for the first time must introduce themselves by saying their name and position or functions they perform within the health team and politely asking for the person's name, if they did not know it previously or if they had not yet said it.

From then on, he will always address her by her name. Sometimes, the person has more than one name and is accustomed to being called by one of them, by the surname or by a nickname. It is advisable to ask her how she prefers or likes to be called, which helps reduce tension and establish better empathy.

Next, the purpose of the meeting must be informed and the approximate time it will take, which also helps to dispel anxieties, doubts and even hostility, and a more rational use of the available time can be made, without digressions unrelated to the defined purpose. The time of the meeting should never exceed one hour and should always be done with the prior consent of the subject.

A common conflict that students present when they begin clinical practice is the feeling of losing the legitimacy of their work; They often feel that they are "playing clinicians" and that the patients they work with could share their discomforts and intimate information with them, as they would with the patient next door, in the office or in the adjacent hospital bed. This idea is reinforced by some comments such as *; "They are so young!"; or the occasional patient who questions the role of clinical students.

Students can handle this situation by explaining from the beginning the degree or level of training and the type of responsibility assigned. For example, "I am a second-year medical student and I am here to perform a physical examination (or interview you) as part of your medical history. Please note that as I develop my technical skills, new information important to your health may be discovered. This should take no more than forty minutes; if you agree, we can begin."

Sometimes, students also have the embarrassing feeling of being "using patients" when they attend to educational objectives, and not their true care, repeating interrogations or physical examinations, which are apparently clinically unnecessary.

However, students should keep in mind that, as stated above, they may discover something important that has not been detected before; that most patients like to have something that is useful for teaching, and that many patients appreciate the extra attention and time that students spend examining them or listening to their stories as beneficial.

Communication during the physical exam

In this stage, as in all stages of communication, it is necessary to achieve the trust of the subject and to do so, they must be reassured and instilled with confidence by previously explaining to them the safety of the examination, that it will cause the least possible discomfort, as well as each subsequent step of the exploration and the need for their cooperation in what they are asked to do, also expressing whether or not they feel discomfort or pain in the area explored, before or during the maneuver.

When communicating with the subject, close attention must be paid to the extraverbal language of both the examiner and the examinee. The person being examined, as we will see in later chapters, can provide many elements of his or her emotional state through extraverbal language. During the physical examination, sometimes a patient may say that he feels no pain, or that it is minimal, when his extraverbal expression shows the opposite;
and sometimes, a malingering or hysterical subject verbally expresses pains and discomforts that are not in accordance with his extraverbal language.

On the other hand, the explorer must also be very careful with his extraverbal language. You should never make gestures that denote impatience, annoyance, boredom, haste, or that could be interpreted as mockery. During the examination, extraverbal expressions should be avoided, contrary to the oral message that, for example, seeks to reassure the subject through words, denying importance to a finding of the examination, after a gesture of surprise, admiration or concern for what was found.

The farewell

Once the physical examination or medical interview is completed, the examiner must thank the examinee for the cooperation provided, the satisfaction of being able to assist him, shake his hand again, and accompany him to the exit.

MEDICAL ETHICS

Ethics is the science of morality, its origin and development, its rules and standards of conduct, duties towards society, the state, or a profession.

Medical ethics is a manifestation of general ethics. In our environment, it is the set of principles and standards that must govern the conduct of health workers, developed in certain specific professional activities, aimed at achieving the preservation and improvement of the health of healthy people and the adequate treatment and recovery of sick people, within a fraternal and human, deeply scientific framework.

According to this, medical ethics should not be considered as a subject similar to the others within the medical curriculum. Ethics being a duty, an obligation of every health worker, it is essential to learn them in each subject and in each rotation, during all medical studies.

ETHICS IN MEDICAL PRACTICE AND PHYSICAL EXAMINATION

Through language, ethical principles that must be taken into account during the interview can be violated, because they affect communication. One of these principles is the treatment of the person, in accordance with the formal education standards of the society in which one lives.

In countries of Latin origin like ours, where the treatment of you and you is well differentiated, it is not correct to address an adult who is older than the doctor or the student. Nor should the members of the health team be addressed on a first-name basis when they are on work duties, including doctors, nurses, technicians and service personnel, even if they are of similar age or have ties of friendship outside of work; The same thing happens with a subject whose social responsibility is known: relevant professional, community leader, religious, state or political leader. Furthermore, any patient who is seen for the first time should not be addressed on a first-name basis, regardless of age, unless it is a child or adolescent.

Get used to calling all people "you" during your work performance as a professional, or as a student, even those of similar age or younger, including all patients, unless you have a strong prior friendship, or the person insists that you call them "you." This, in addition to demonstrating formal education, denotes maturity, seriousness and a sense of responsibility in your work, which compensates for your youth and is reciprocated with respect for others and treatment in line with his role as a health worker and not as just another student or a simple "young doctor."

Nor should expressions be used to address a patient, such as "uncle" or "grandfather", which, although they are commonly used by the new generations in our environment, denote rudeness, are disrespectful and can even be offensive, mainly in middle-aged people.

For optimal compliance with medical ethics in healthcare institutions, from the office to the highest level research institute, one must speak quietly and demand the same from others; There must also be sufficient privacy, and without unnecessary interruptions, both for the interview and for the physical examination. When the latter is performed, the doctor, since he is a student, must get used to being careful and courteous, especially with the elderly, helping them, for example, to lie down or get up on the stretcher. At all times you must keep in mind respect for modesty, especially with women, using curtains or a paraban, if necessary, and keeping covered the parts that are not essential to be exposed for the examination being carried out. Always remember to explain in advance everything that is going to be done and reassure the patient, to achieve the greatest cooperation and avoid tension and anxiety, especially when the breast, genital, and gynecological and rectal examinations are to be performed. It is recommended to be accompanied by the nurse, a close family member or another member of the team, if the person to be examined is young and of the opposite sex to that of the examiner, to avoid situations that are embarrassing for both or that could be interpreted as a violation of medical ethics.

In relation to teamwork and medical ethics, we wanted to leave for last, not because they are less important, but on the contrary, two essential ethical issues in medical work: respect for the hierarchical level and the interpersonal relationships of the health team, during the exercise of their functions, whether in front of the patients or not.

Respect for the hierarchical level is essential in medical practice. It includes respect and obedience to students in higher years, such as boarders, even if they are of similar age, as well as residents and non-teaching specialists. Sometimes, because the students and interns are the youngest within the hierarchical organization of the health team, they play with them and among themselves and treat each other with the familiarity typical of young people; But this type of interpersonal relationship, in the midst of work and educational activity, and in front of patients, goes against their ethical training as professionals and the medical ethics of the team, since the majority of patients question whether their health will not be in the hands of people who can treat their problem with the same lack of seriousness and response.

responsibility they show in their interpersonal relationships.

On the other hand, because they are the lowest hierarchical echelons and have less experience, they are sometimes underestimated by the intermediate hierarchical staff and their role within the health team is not taken into account or taught with respect, which, together with overconfidence, when the demand for work completion is required at a given time, the hierarchy is not respected and attempts are made to impose it by force, which creates a conflict in team relationships, which is generally unleashes in front of patients.
Similar conflicts over who should do certain work with the patient can occur among the students themselves.

The same happens with the relationships of students with nursing students and professionals, and with paramedical personnel, such as technicians, secretaries, cleaning staff, etc., in which the daily lack of mutual respect is interpreted as such, when the conflict is created, because one of the parties demands responsibilities from the other, then the respect that they had not earned is demanded and the hierarchy now wants to impose itself.

These last negative examples constitute multiple violations of medical ethics, because not only is the patient at the center of them, but there may be violations of different complexity among the members of the team who intervene in it.

The student, therefore, must learn from the beginning in clinical work that problems, even if they are work-related and whether or not they are directly related to patient care, whether among the students themselves or with another member of the team or health worker, must be discussed alone, away from the presence of the patients.

Other violations of medical ethics that also constitute iatrogenesis are the negative attitudes of the health team when performing work functions, such as commenting on news, telling television programs or having conversations about domestic or personal situations such as parties, etc. unrelated to medical care, when a patient is being consulted, some procedure is performed, or the subject is waiting to be treated. The person feels that they are not given the attention they deserve and fear the consequences that this unserious and irresponsible attitude may have on their state of health.

IATROGENIC

The iatrogenic disorder (from the Greek iatros: doctor; genus: origin) is that which is caused by the doctor himself, or in a broader sense, that which derives from medical care.

Iatrogenesis can be psychological, when it damages the psychological integrity of the individual and their ethical-moral principles, which can even lead to the aggravation of organic diseases; and it can be non-psychological, when it damages the physical integrity of the individual; For example, due to the administration of a medication, or due to a poorly performed or excessive operation, etc.

In Chapter 22 of this work, the main mechanisms involved in the iatrogenic process are briefly explained.

In clinical practice, errors in communication and violation of ethics are causes of iatrogenesis and, in turn, committing iatrogenesis of any type is a violation of medical ethics that can even have legal implications of great significance.

As iatrogenesis due to defects in communication, we can mention those produced through language, both verbal and extraverbal, some of which have already been mentioned in this chapter.

One of the issues that can cause verbal iatrogenesis is the use of technical language in communication, which does not allow the subject to adequately understand the meaning of the message and interpret it incorrectly, aggravating their state of health. On other occasions, the doctor or the student gives lectures in front of a patient, without reassuring him or her beforehand clarifying that they are purely academic and unrelated to his person or his state of health; The same thing happens when faced with a finding during a physical examination or in an investigation, sometimes banal, that with the doctor's desire to teach, or out of eagerness or demonstration of knowledge on the part of the student, they express in the presence of the patient, everything theoretically related to the finding; even, to the prognosis or complications that may arise from it.
give rise.

Or it may happen that, both the student and the professional, in order for the patient to know their diagnostic skill and competence, tell the person: "you have a murmur" or "you have a right bundle branch block", with the lack of tact and without the due prudence to explain in both cases, that the finding can be found in people without heart disease and has no impact on their state of health, although it may be necessary. investigate it better for greater peace of mind.

Another issue related to iatrogenesis through verbal language is the use with or in front of the patient of medical and non-medical terms that patients can dominate or reject socially. In the first case we have, for example, the use of the word " tumor" or "tumor", which in the medical language of the

physical examination, is synonymous with *increase in volume".

but which in popular language means "cancer". Likewise, in our environment the population knows the meaning of " neoplasia" (which it always interprets as malignant), " leukemia", cirrhosis" and many others, such as "dementia" and "hysteria", or misinterprets "atherosclerosis" as dementia".

As for the words that are socially rejected and that no one likes to be handled openly, they are "tuberculosis." , which is replaced by TB", "drunk or "alcoholic", which are replaced by "ethyl" and "chronic alcoholism", respectively; "syphilis and syphilitic", for "lúes and luético"; "leprosy and leper", for "Hansen's disease and Hansenian", respectively; and many others that will be learned progressively in medical practice, but that cause iatrogenesis, if care is not taken with language, during medical practice.

Performing maneuvers in a sudden manner that causes unnecessary discomfort or pain, not respecting modesty or creating embarrassing situations for the subject are also an important cause of iatrogenicity during the physical examination, as previously addressed in the topic of "Medical Ethics."

It would be illusory and it does not correspond to our objectives to want to exhaust the topic, although we would like to end this chapter by remembering that since ancient times there has been a concern that the doctor, far from curing or improving the patient, achieves the opposite, as witnessed by the classic Latin apothegm Primum non nocere. (First, do no harm.)

2 THE PHYSICAL EXAMINATION AND ITS BASIC EXAMINATION

The physical examination is the examination that the doctor personally performs on every individual, in order to recognize the existence or not of physical alterations or signs produced by disease, using only the senses and small devices carried with oneself, such as the clinical thermometer, the stethoscope and the sphygmomanometer, to mention the most common.

The four basic techniques of clinical examination are: inspection, palpation, percussion and auscultation.

INSPECTION

Inspection is the appreciation with naked eyes or at most with the help of a magnifying lens, of the characteristics of the body on its external surface and of some cavities or ducts accessible by their extensive external communication, for example, mouth and fauces.

When the physical examination is carried out as such, the examinee will stand, sit or lie down, according to what we want to examine and with the physical or facultative limitations that he or she may have, and the examiner will stand in front of him, with his back to the light, if the person being examined is standing or sitting, or on the right side if he is lying down, when the explorer is right-handed, and on the opposite side when the person being examined is left-handed.

INSPECTION TECHNIQUE

General guidelines. Both in direct or immediate inspection and in mediate or instrumental inspection, appropriate lighting is essential, whether natural or solar, or artificial. Whenever possible, natural light should be preferred, especially reflected or diffuse light, since with artificial light, necessary in some inspection techniques, certain colors, such as red, may undergo modifications, and others, such as yellow, may go unnoticed. Thus, if natural light is not used, the existence of jaundice could be unknown because the yellow color of the skin and mucous membranes that characterizes it cannot be identified.

Execution. The inspection is carried out at all times, even before beginning the physical examination itself, from the moment we see the individual, until our communication with him ends. For your axis-

cution as an operation the following invariants should always be considered:

- Appearance and/or symmetry.
-Color.
- Shape.
- Size.
- Mobility.

PALPATION

Like inspection, palpation is one of the oldest procedures in the physical examination. Susruta, a Hindu doctor from the 5th century, routinely palpated the pulse, but it was Francisco Hipólito Albertini (1726) who systematically practiced it for the diagnosis of chest diseases.

Palpation is the manual appreciation of the sensitivity, temperature, consistency, shape, size, location and movements of the explored region, thanks to the exquisite tactile, thermal, vibratory, pressure and stereognostic senses of the hands.

This assessment is carried out in the integuments or through them, and then it is simply called palpation, or by introducing one or more fingers and even the hand through natural channels, and then it is called touch, for example, rectal and vaginal touch.

PALPATION TECHNIQUE

General guidelines. Palpation of the integuments is carried out with the bare hand, except for possible contamination. Instead, touching is done with a protected hand, either with fingers or rubber gloves, lubricated to facilitate penetration.

Execution. Palpation can be monomanual or bimanual; that is, with one hand or with both, either by having to use the latter separately, superimposed or juxtaposed. It can also be digital, if only the use of one or several fingers is required, as in palpation of the neck or pulses.

For its execution as a palpatory operation of any structure, throughout the entire physical examination, the following invariants must always be considered:

- Situation.
-Shape.
- Size.
-Consistency.
Sensitivity (pain and temperature).
- Mobility.

PERCUSSION

It consists of the appreciation by the ear of the acoustic phenomena, generally noises, that originate when the external surface of the body is struck.

This method was invented by Leopold Joseph Auenbrugger in the mid-17th century and led to enormous progress in the diagnosis of chest diseases, but since the advent of Roentgen rays, it has lost much of its value, since both provide analogous information, radiology being much more exact, so much so that it has been said that the errors of percussion are centimeters, while those of radiology are only millimeters.

PERCUSSION TECHNIQUE

General guidelines. Percussion can be practiced by hitting the external surface of the body, with bare hands or using a special instrument called a percussion hammer.
Hammer percussion will be explained in the chapter " Exploration of the nervous system" and manual percussion can be digit-digital, digital or fist percussion.
In digital percussion the percussing finger strikes directly on the surface of the explored area, it is the least used, it is practiced, for example, on the skull and some other bones and on the muscles, looking for abnormal contractions, while in digital-digital percussion the percussive finger strikes on another interposed finger (plesimeter finger), located on the surface that is percussed; It is the most practiced, because it creates the purest percussion noise, because it is performed in any region, and, furthermore, because it provides a sensation of resistance in the interposed finger which, added to the acoustic sensation, contributes to a better appreciation.
Fist percussion is characterized by hitting the ulnar edge of the closed hand or fist, generally on the back of the other interposed hand. It is practiced especially in the lumbar region to detect the pain caused by inflammatory conditions of the kidney. Its technique will be explained in the chapter on " Exploration of the urogenital system".

Execution. The digit-digital percussion technique requires some important points and systematic practice, if we want to obtain value from its use.

The interposed finger or pleximeter finger can be the middle or index finger, supported enough to establish intimate contact of only the palmar aspect of the second and third phalanges with the wall. To do this, these phalanges must be fixed with a slight hyperextension of the finger that does not allow the first phalanx to touch the surface. On the other hand, while it is indifferent

Use the pleximeter finger in any direction, that is, with its long axis perpendicular, parallel or oblique to the midline. On the thorax, orientation parallel to and over the intercostal spaces is preferable, and not over the ribs.

As for the striking hand, the index finger, the middle finger or both can be used, but with care to place it or them in a semi-flexed position, so that the distal edge of the finger hits perpendicularly on the phalanges or the distal interphalangeal joint of the plesimeter finger.

At the same time, the wrist must perform extension and flexion movements successively, accompanied by a very slight swing of the forearm and arm. Only two successive blows should be given in the same place, so that the perception obtained can be compared with that of another area. That is, the sequence of the operation will be:

1. Properly position the pleximeter finger.
2. Place the striking finger in position, lightly touching the place where it is going to be hit.
3. Extension and flexion, extension and flexion of the wrist, for the first and second strikes, respectively.

Note that the percussion finger ends practically on top of the pleximeter finger, in an adequate position to carry out another sequence by simply moving the latter. Do not try to make another final extension movement, as if the hammer finger were running away from the pleximeter finger, because this reduces the fluidity of the percussion and hinders your technique.

On the other hand, the types of sonorities obtained according to their tone, from highest to lowest, can be classified as: tympanyism, hypersonority, normal sonority, submatidity and dullness. The sound obtained when striking an object filled with air, such as a mattress or a rubber pillow, is tympanic, while striking a solid object such as wood, a bone, or a thigh is considered dull. However, the timbre of the sound to be evaluated varies from one place to another, and what is considered

Normal sonority in the abdomen may be hypersonorous in the thorax, as will be detailed later in the exploration of each area.

Finally, we dedicate a few words of warning to the students:

They should not hit with long nails, at least that of the striker finger, because doing so can damage the pleximeter finger with the nail, or when feeling pain, it is not hit with enough force or it could be hit with the ball of the finger, which would lose the inestimable value of hitting vertically (fig. 2.1).

They should also practice this exercise a lot to achieve wrist flexibility and to do so they can hit the muscles, bones, furniture in the room and practice with their peers and family.

Algorithm for the execution of digit-digital percussion.

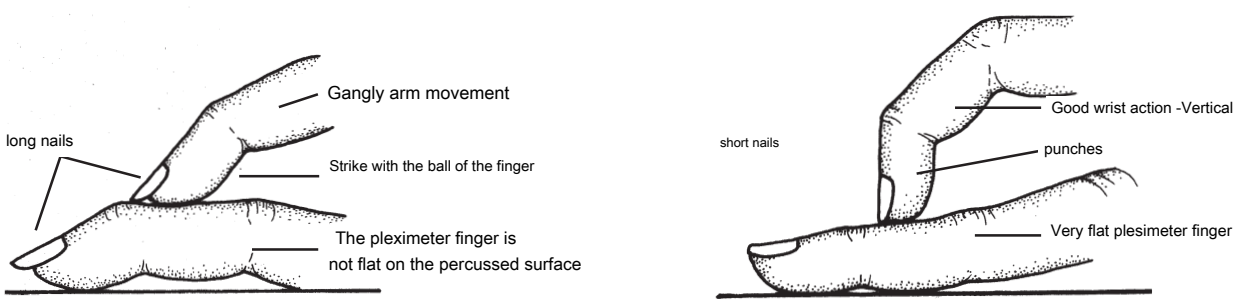
- Proper position of the pleximeter finger.
- Proper position of the striking finger.
- Wrist extension and flexion movements with slight swinging of the forearm and arm.
  Strike perpendicularly with the distal edge of the finger, without a long nail.
- Give only two blows in the same place.
- Evaluate the type of loudness obtained.

It is fair to clarify that there is an alternative percussion for female explorers, who find it difficult to have their nails short enough to perform adequate percussion.

The technique consists of striking with the outer edge of the thumb, creating a pronation movement of the wrist of the striking hand.

Although it is controversial whether the sound and percussive sensation obtained have their disadvantages, compared to those of the classical technique, the prestigious Cuban professor Dr. Mercedes Batule has used it successfully throughout her professional and educational activity.

Fig. 2.1 Percussion method: a, wrong; b, correct.


HOW IT SHOULD NOT BE HIT
Gangly arm movement
long nails
Strike with the ball of the finger
The pleximeter finger is
not flat on the percussed surface
HOW IT SHOULD BE HIT
Good wrist action -Vertical
short nails
punches
Very flat plesimeter finger

When performing digital percussion, the same movement technique and position of the finger of the percussing hand is used as in classic digit-digital percussion.

AUSCULTATION

The discovery of auscultation and the invention of the stethoscope at the beginning of the 19th century by René Laennec, caused a true revolution in the diagnosis of diseases of the thorax (lungs and heart), since a series of pathological conditions unrecognizable in life were easily individualized by this method of examination; for example: bronchitis, pulmonary emphysema, pneumonia, pleurisy, heart valve lesions, pericarditis, etc.

Despite the time that has passed and the advent of other modern methods with a more or less similar purpose, such as the graphic recording of acoustic phenomena, auscultation continues to be one of the fundamental methods of the physical examination of the cardiovascular and respiratory systems.

Auscultation consists of the appreciation, with the sense of hearing, of the acoustic phenomena that originate in the organism, either due to the activity of the heart (cardiovascular auscultation), or due to the entry and exit of air in the respiratory system (pulmonary auscultation), or due to transit in the digestive tract (abdominal auscultation), or finally due to any other cause (fetal auscultation, joint auscultation, etc.).

In other words, three factors intervene in auscultation: the receiving organ of hearing, the nature and characteristics of the auscultable acoustic phenomena, and the technical methods used to listen.

There are two methods for auscultation: immediate auscultation and mediate auscultation.

Immediate auscultation

It is performed by applying the finger directly against the skin surface, generally with the sole interposition of a fine cotton fabric or thread and never silk or other fabric that can generate noise. Sufficient pressure must be applied so that the pinna of the ear adapts around its entire contour, forming a closed cavity.

Mediate auscultation

It is carried out by placing a small instrument called a stethoscope between the ear and the skin surface, which adapts perfectly to the external auditory canal and the skin in the region. The stethoscope can be monaural, such as those used in obstetrics to auscultate the fetal focus, or binaural, such as those used to auscultate the different systems.

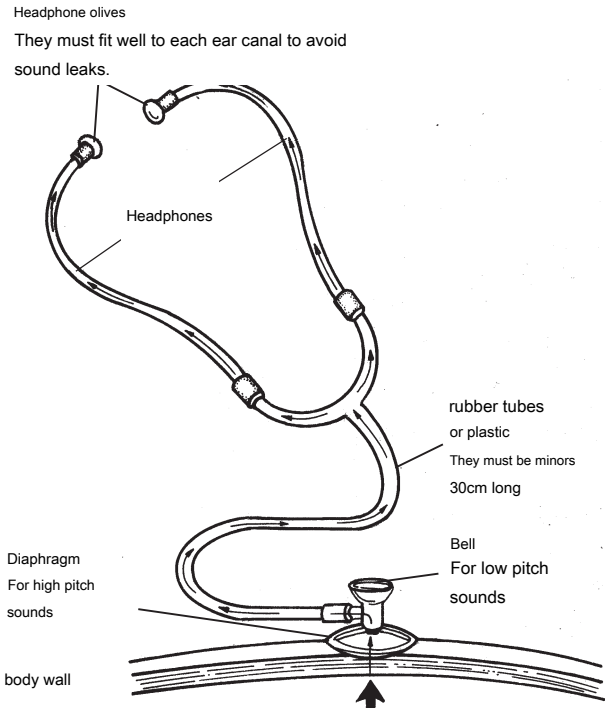


Fig. 2.2 Binaural stethoscope.

Auscultation techniques will be detailed in the chapters dedicated to these systems, especially the one dedicated to cardiovascular examination.

binaural stethoscope

It is the most common instrument used to transmit heart sounds from the rib cage to the ear (fig. 2.2).

There are several types of stethoscopes, and it is essential to have a basic knowledge of the properties of each one. Since these have certain characteristics, it is advisable to become familiar with a specific type and use it most of the time.

Headphone parts. For the sound to be transmitted through the stethoscope, the system must be airtight, since any continuity solution greatly attenuates the sounds. Therefore, it is important that the earpieces are of appropriate size and shape and that they fit well in the ear; The axis that joins both ear pieces must be elastic enough to keep them firmly in place.

Rubber tubes. The shorter the tube system, the more efficient the stethoscope will be. By increasing the length of those, the fidelity of the stethoscope to transmit high-frequency sounds (above 100 cycles per second) decreases.

Receivers. There are two basic types of receivers, bell-shaped and diaphragm. The first type may have

different shapes and sizes. The larger the diameter of the bell, the more easily low-pitched sounds are transmitted. However, it is difficult to place the large diameter bell on the chest of thin patients and children. The 2.5 cm diameter hood is more widely accepted, the size of which is adequate and small enough to fit exactly to the wall.

Used correctly, the bell receiver is best for picking up low-pitched sounds.

The Bowles-type receiver has a rigid bakelite diaphragm. It is of variable size, but the most common is 3.9 cm in diameter. It has a relatively high natural frequency that reinforces high-frequency sounds, but it does so with an overall loss of sensitivity, especially notable in lower-frequency sounds.

This receiver is suitable for auscultating murmurs and high-pitched noises. Comparing the bell receiver with the diaphragm receiver, the loss of sensitivity of the latter is compensated by the advantage of its larger diameter.

Most of the time, when auscultating, both types of receivers must be used: the bell, for general auscultation and for lower frequencies, and the diaphragm for higher frequencies.

Almost all modern stethoscopes have receivers that combine the bell and diaphragm. The Rappaport and Libman models have been perfected, and they have quite a bit of fidelity.

amplifier stethoscope

It has a definite value for certain patients and for some doctors. It is useful for people with poor hearing, but should not in any way replace thus the silent environment essential for auscultation. Most significant murmurs can be heard without the need for an amplifier. Amplifying stethoscopes with filters are very useful for personal training or teaching. With any of the amplification systems, the sound can be very different from that commonly heard with the binaural stethoscope. The doctor must know his device well and be able to differentiate the artifacts inherent to the instrument from those that result from technical and mechanical problems.

AUSCULTATION TECHNIQUE

When performing auscultation as an operation, the following should be considered:

-Correct placement of the earphone when auscultating.

- Hold the diaphragm or bell with two fingers (thumb and index or middle finger).

-Heat the diaphragm by friction if necessary.

- Apply the diaphragm firmly to hear sharp or loud sounds.

- Apply the bell gently to hear low or low sounds.

The characteristics of the sounds to be taken into account during auscultation, such as intensity, tone, timbre and others, will be discussed in the chapter on the examination of the cardiovascular system, in this same Section.

To conclude, it is necessary to clarify that not all techniques include the four basic exploration methods. For example, in the examination of psychological aspects, attitude and gait, we only use the inspection technique.

GENERAL PHYSICAL EXAMINATION.

YOUR EXPLORATION TECHNIQUES

3

The complete physical examination, from a didactic and written record point of view, consists of three parts:

1. General physical examination.
2. Regional physical examination.
3. Physical examination by systems.

In this chapter we will explain the techniques of the general physical examination, which in turn are subdivided into the exploration of:

- Gait, biotype, attitude and facies.
-Size, weight and temperature.
- Skin, mucous membranes and skin (hair and nails).
- Subcutaneous cellular tissue and adipose panniculus.

GAIT, BIOTYPE, ATTITUDE AND FACIES

During the inspection, from the first moment of meeting the person and establishing communication, these details must be observed, separately and as a whole, to allow conclusions to be reached.

MARCH

The first of the data that we collect through the general inspection is whether the individual walks more or less freely, or if, on the contrary, he is limited, to a greater or lesser extent, or forced to remain seated or in bed, when the examination is carried out at home or in the hospital. Observe the person's movements, upon entry into the room, to assess gross movements in gait and posture. You should also evaluate whether the person experiences difficulty or pain when standing or sitting, whether or not to later perform a more specific examination.

It must also specify whether there are abnormal movements that may indicate dysfunction (tics, tremors, spasticity, etc.), which will be studied in the Clinical Propedeutics Section.

For a more specific exam, ask the person to walk to detail the motor function of gross movements (walk, stance, stay) and the range of motion of the joints used for walking.

Normal gait is smooth, usually accompanied by symmetrical swinging of the arms. When walking is normal, the heel should gently touch the ground with the knee extended. The step must then be transferred harmoniously along the foot towards the metatarsal. With the knee slightly bent, the foot should be lifted off the ground.

Walking movements must be coordinated.

BIOTYPE OR EXTERNAL HABIT

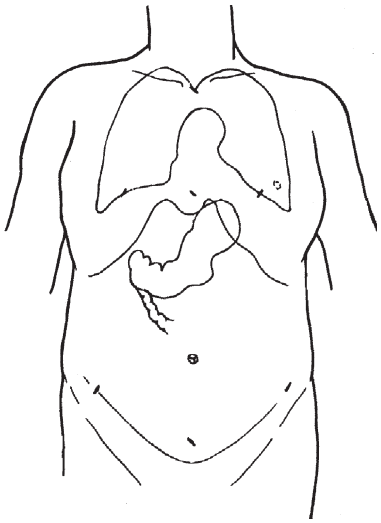
Let us now proceed to study, also through general inspection, the body conformation of the subject, its external habit, which leads us to confront the important problem of individual constitution. At this moment we will only make a brief allusion to this transcendental issue to which, due to its importance, we will devote a special study in the Clinical Propedeutics Section.

If we carefully observe the body conformation or external habit of the different individuals, we will see that they can be separated into three large groups: Some are generally short in stature, are rather thick and corpulent, have a short neck, wide thorax, a very open, obtuse epigastric costal angle, well-developed musculature and relatively small limbs: they are the brevilineal, megaloplanchnic (from the Greek megas: large; esplangkhnon: viscera), pycnic (from the Greek pyknus: thick, condensed) or hypersthenic (from the Greek hyper: excess; sthenos: strength) (fig. 3.1). Others are, generally, taller, thinner, with an elongated neck and thorax, a closed and acute epigastric costal angle, poor musculature and long limbs: they are longilinear, microsplanchnic or asthenic (from the Greek a: privative;

sthenos: strength) (fig. 3.2).

Finally, the normolinear, normosplanchnic or sthenic, constitute the intermediate group with a harmonious body conformation (fig. 3.3).

Fig. 3.1 Briefline type.



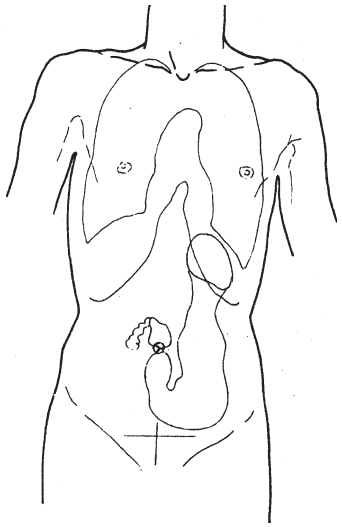


Fig. 3.2 Longilinear type.

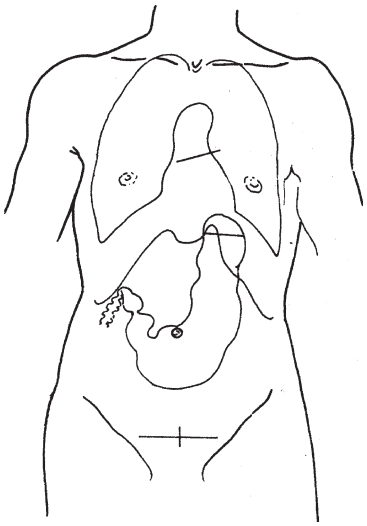
Such a markedly different typology suggests, as we will later confirm in Chapter 24 of Section II, a different ease or predisposition to contracting diseases, as well as an unequal resistance to them.

ATTITUDE

The posture, attitude or stay refers to the position that the individual assumes when standing or sitting and also lying down.

The attitude adopted when the person is lying down (called " attitude in bed) is variable within normality and its importance lies in the pathological attitudes that a bedridden patient can assume, so its examination will be described in the Clinical Propedeutics Section.

Fig. 3.3 Normolinear type.



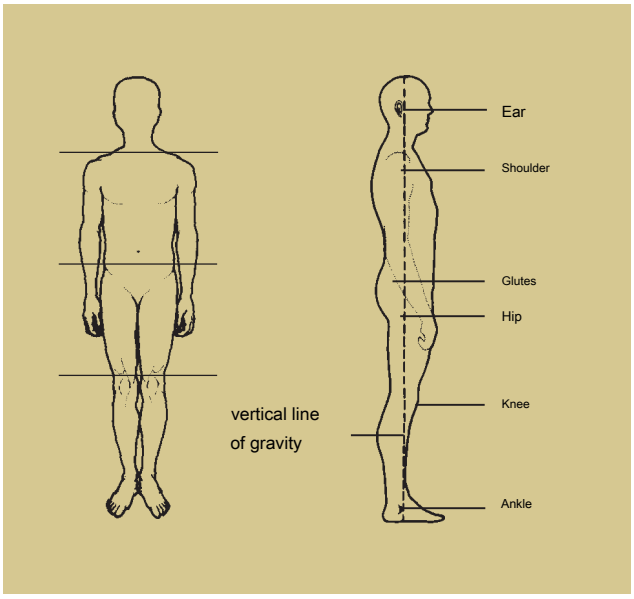


Fig. 3.4 Normal alignment in the standing attitude: a, facing forward; b, profile.

Standing attitude. Good posture or normal standing attitude is characterized by proper alignment of body parts.

To do this, ask the person to stand in front of you and, at a distance of about 2 m, evaluate the alignment and symmetry of the shoulders, iliac crests and knees, which should be at the same level, respectively. Then ask him to stand in profile. The alignment is normal if in the lateral position an imaginary line can be drawn that passes through the earlobe, shoulder, hip, femoral trochanter, the center of the knee and in front of the ankle (Fig. 3.4).

In this same position, the type of posture can also be evaluated, based on the Classification of Postural Types, by Lloyd T. Brown, obtained by him in a study carried out on 700 students at Harvard University, which evaluates the appearance of the head, thorax, abdomen and the curvature of the back.

The postural types are the following (fig. 3.5):

Type A: excellent

1. Head erect. Protruding chin. Perfect alignment of the head in relation to the shoulder, hip and ankle.
2. High and domed chest. The sternum is the most anterior part of the body.
3. Lower abdomen inward and flat.
4. The curvatures of the back are within normal limits.

Type B: good

1. Head slightly forward.
2. Slightly low chest.
3. Lower abdomen inward, but not flat.
4. Slightly increased curvatures of the back.

Type C: poor

1. Head forward.
2. Flat chest.
3. Relaxed abdomen. It is the most promising part of the body.

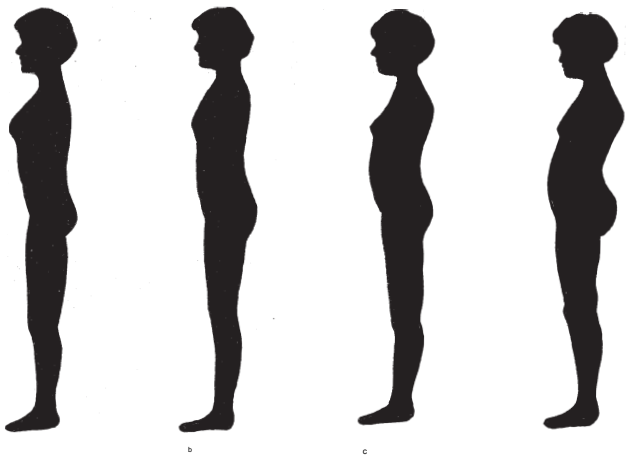
nent.

4. Exaggerated curvatures of the back.

Type D: bad

1. Head exaggeratedly forward.
2. Depressed (buried) chest.
3. Abdomen completely relaxed and protruding.
4. Extremely exaggerated curvatures of the back.

Fig. 3.5 Postural types: a, excellent; b, good; c, poor; d, bad.



b c

In general, normolineans adopt type A as their postural type; the brevilinear ones, type D, and the longilinear ones, the Bo C type.

Deviations from good posture can be influenced by habits (prolonged standing, slumping of the shoulders or sitting in an inclined position).

Abnormal postures are generally due to neurological or musculoskeletal diseases and other systems (for example: respiratory, cardiovascular).

A more thorough examination of the standing attitude is performed during the examination of the osteomyoarticular system. For more details see chapters 7 and 13 of this Section.

FACIES

This is what the appearance and configuration of the face, the facial or physiognomic expression of the person is called.

First of all, we must evaluate facial symmetry, both static and in movement, comparing the right side.

of the face with the left, evaluating the wrinkles of the forehead, eyebrows, eyelids, eyes, nasolabial folds and the corners of the mouth.

If there is no asymmetry, a wide look, opening the eyes completely when speaking, can express anxiety; Frowning and narrowing the eyelids denotes displeasure; An evasive look can denote shyness or sadness.

The facial expression that we collect in the written facies record can be, among others: tense or anxious, questioning, angry, happy, sad, painful, inexpressive, etc., although its objective description is preferable, followed by the meaning that in our opinion it denotes.

Mood states such as anxiety, disgust or anger and sadness are detected not only by facial expression, but also by the way of walking, posture and the way of speaking.

Sadness manifests itself with a slow and painful step when walking, dropping the head, slumping the shoulders, indifferent posture and evasive gaze. When he speaks he does so at the final point of inspiration.

The subject with disgust or anger has a lively and energetic gait, when sitting he crosses and moves his legs or stretches them, clenches his fists, frowns, looks defiantly and speaks forcefully during exhalation.

Anxiety, for its part, is manifested by a fast, but unsteady step; posture is rigid when sitting, rubs hands or wipes sweat; bitten nails:; wide look, opening the eyes; and speaks at the highest point of inspiration.

SIZE, WEIGHT AND TEMPERATURE

Weight and height are the anthropometric measurements that must be examined in the physical examination of every person. This is part of the parameters necessary to evaluate the nutritional and metabolic status of the individual.

The equipment necessary to carry out these measurements consists of: a tape measure and a scale or weight, preferably with a bar scale, and which may or may not have a stadiometer incorporated.

SIZE

Height recording in young adults can be done once, but it is necessary to examine it periodically from birth to adolescence, to evaluate growth, as well as in elderly people, to evaluate shortening due to narrowing of the intervertebral discs or compression fractures.

Height is also used to estimate ideal weight and interpret other data or determine other values such as body architecture and body surface area.

To take it, ask the person to stand erect, without shoes, with their back to the wall where a tape measure has been fixed or drawn, or to the stadiometer of the scale.

Make sure your feet are joined at the heels and that These, the buttocks, shoulders and head, are touching the wall or the height rod. Record the height measurement in centimeters. If the scale is in inches, convert it to centimeters by multiplying the figure by 2.5.

Using the scale's stadiometer to measure height is less safe than the tape, but safer than asking the person how tall they are.

If the examinee cannot stand, measure the height in a supine position, with the body fully extended, with a tape measure, from the heels to the end of the head.

WEIGHT

Weight must be measured and recorded at all matches. The fact of gaining or losing weight should be an important indicator of health or illness. Often, the dosage of medications depends on weight.

Some authors accept as a general rule for calculating the weight that should normally correspond to an individual (ideal weight), the following: the ideal weight is equal to as many kilograms as there are centimeters left over 100 in the subject's height (Broca's Formula), admitting a margin of normal deviation of up to 10 kg and specifying that the weight should be somewhat lower in women.

Standardized weight tables recommend ideal weight values, according to height, age, sex and body architecture or build, which can be small, medium and large (tables 3.1, 3.2 and 3.3).

Table 3. 1 Normal weight and height. Age: 6 months to 20 years

Age (in years)	Men		Females	
	Size (in cm)	Weight (in kg)	Size (in cm)	Weight (in kg)
	66	7.7	66	7.1
1	74	9.5	76	9.0
2	84	11.8	84	11.3
	92	14.0	92	13.6
4	99	15.9	99	15.4
5	107	17.2	104	16.8
6	115	19.5	112	19.5
7	120	22.6	120	21.3
	125	25.0	125	24.5
	129	27.7	129	27.2
10	135	30.4	135	30.4
11	139	34.0	139	33.6
12	145	36.8	145	37.2
13	150	40.9	152	42.7
14	158	46.8	158	47.7
15	162	50.8	160	50.8
16	168	57.2	162	53.1
17	170	60.4	162	55.4
18	172	62.7	165	56.3
19	175	62.7	165	57.2
20	175	63.2	165	57.2

Table 3.2 Ideal weight for women. Age: 25 years and older

Size (cm) (with shoes)	Constitution and weight (kg) (usually dressed)		
	Small	Median	Big
150	47.3-50.4	49.9-53.6	53.1-57.5
152	47.7-51.3 50.8-54.4		54.0-58.6
155	48.6-52.2 51.7-55.4		55.0-59.5
158	49.9-53.6	53.1-56.8	56.3 61.3
160	51.3-55.0	54.5-58.1	57.7-62.7
162	52.7-56.8	56.3-60.0	59.5-64.5
165	54.0-58.1 57.7-61.3		60.4-65.8
168	55.9-59.9 59.1-63.8		62.7-68.3
170	57.2-61.7 60.9-65.4		64.5-69.9
172	58.6-63.2	62.2-66.7	65.8-71.8
175	60.4-65.0	64.2-68.7	67.6-73.6
178	61.7-66.7	65.8-70.3	69.1-75.3
181	63.1-68.3 67.2-71.7 64.2-69.5 68.		70.3-76.8
183	7-74.1		72.7-79.1

Table 3.3 Ideal weight for men. Age: 25 years and older

Size (cm) (with shoes)	Constitution and weight (kg) (usually dressed)		
	Small	Median	Big
158	52.7-56.8	56.3-60.4 59.5-64.5 60.4-65.4	
160	54.0-58.1 57.7-61.7		59.0-63.7
162	55.4-60.0	62.2-67.7 60.9-65.4 64.2-69.5	
165	57.2-61.7	62.2-66.7 65.8-71.2 64.1-68.7	
168	58.6-63.1	67.6-73.5 65.8-70.9 69.9-75.4	
170	60.4 65.0	67.6-72.7 71.3-77.2 69.5-74.5	
172	61.7-66.7	73.2-79.5 67.2-72.1 71.3-76.3	
175	63.8-68.7 65.	75.0-81.7	
178	4-70.4		
181			
183	69.0-74.5 73.7-78.6		76.8-84.0
185	71.3-76.8	75.4-80.9	79.1-86.5
188	74.1-79.5	77.7-53.6	81.3-89.2 83.
191	76.3-82.0 80.0-85.9		6-91.8

The body architecture or build is obtained by dividing the size by the circumference of the wrist, also in centimeters.

Wrist circumference is measured by placing the tape measure around the wrist where it joins the styloid process.

Size/Wrist circumference Women Men		Body build
> 10.4 9.	> 11	Small
6-10.4 < 9.	10.1-11 <	Medium
6	10.1	Large

To proceed with weighing, follow the guidelines below.

tes:

1. Obtain the weight on a scale, preferably an arm scale (arm weights are safer than bathroom weights), with the individual barefoot, with as little clothing as possible, after emptying the bladder and if possible, after defecation, but never after eating.

There are bed weights for immobile patients.

2. Record the weight in kilograms.

If the scale is in pounds, do the dividing conversion.

do these by 2.2. A weight greater than 10%, above or below the ideal weight is associated with nutritional dysfunction.

3. Record next to the current weight, the usual weight you

determines the person and the ideal weight, according to the size.

4. Standardize the procedure as much as possible. Serial weighing, such as daily weighing, during hospital admissions, should be obtained at the same time each day and using the same amount of clothing.

BODY TEMPERATURE MEASUREMENT

Whether or not a person has an increase in body temperature is suspected by palpating their skin with the back of the hand in different regions: forehead, neck, trunk, extremities.

But body temperature is determined by reading the measurement recorded on a thermometer, which can be the classic clinical glass thermometer, which contains mercury (Hg) inside, an electronic thermometer or using a monitor associated with a thermodilution thermostat, such as the one that accompanies a catheter implanted in the pulmonary artery or a Foley catheter.

Clinical glass thermometers are called maximum thermometers, because when the mercury rises to the maximum marked temperature, it remains there until it is lowered mechanically.

Electronic thermometers are the safest and are preferred over glass ones because they have less chance of cross contamination due to the disposable cover used for each intake.

For a reliable reading, these instruments must be used and read correctly.

The scale of the thermometers is variable. The most universally accepted and used in Cuba is the Celsius or degrees Celsius (°C), in which the thermometer is divided into degrees and tenths of these. They are only marked at 34-42 °C, which are the limits that normal clinical needs require. In the Saxon countries, the Fahrenheit (°F) scale is used, which must be known, since in some cases of clinical temperature examination (Tomkins test or thermal curve),

monthly basal mica in women) is advisable given that small variations are more noticeable in her.

The correspondence between both scales is easy to know, taking into account that the 100 degrees of the centigrade scale (100 °C) correspond to 180 degrees of the Fahrenheit scale (180 ° F), since the latter extends from 0 °F to 212 °F but 0 of the centigrade is equivalent to 32 ° of the Fahrenheit.

To convert degrees Fahrenheit (°F) to Celsius or centigrade (°C) we solve:

Temperature in °F-32. 5 9

And to convert degrees Celsius (°C) to Fahrenheit (°F):

____ T(°C) -9 5 ____ +32

The fraction 5 6 9 arises from the equivalence 5

_ 100 or 180 between degrees Celsius and Fahrenheit.
180 100

The temperature can be taken in different places on the body, such as the mouth, rectum, armpit, inguinal region, and vagina.

Armpits. The axillary temperature is the most used in Cuba, but its practice should be abandoned due to its inaccuracy, and replaced by the oral temperature. The axillary method is preferred for children because it is less dangerous than the other methods.

In the armpit, prior to thoroughly checking its dryness, the thermometer should be left for at least 5-10 minutes.

Mouth. Oral temperature, the most widespread in the Saxon countries, is more accurate than the previous one and should be used every time the doctor wants to know with certainty what the subject's real temperature is. The thermometer should be left in the mouth for about 5 minutes, although the manufacturer may indicate less time. It requires a thermometer for each patient in hospital centers and for each person while they are sick at home. The thermometer should be washed and kept in 70° alcohol for around 10 min.

The oral method can be used in people who are alert, alert, cooperative, and over 6 years of age. The person must be able to breathe through the nose and must not have any oral disease or recent oral surgery.

tea

Straight. The rectal temperature, widely used in Europe, is considered the most accurate of all, but also the most invasive, uncomfortable and embarrassing, which is why it is rarely used. in Cuba. Requires special rectal thermometers. It should be used in children under 6 years of age or in anyone in a confused state, prone to seizure activity, in those who are comatose or intubated. Some authors consider that rectal thermometers should not be inserted in children under 2 years of age, due to the possibility of rectal perforation. It is also contraindicated after some operations, such as hemorrhoid surgery and abdominoperineal resection, and in people with heart disease, because rectal stimulation can cause Valsalva maneuver-type effects.

Its use is essential when the person is being subjected to a therapeutic hypothermia process. For the latter, there are electronic thermometers, for recording and graphically recording the temperature.

Groin. Inguinal temperature is used especially in children and very weakened patients; It has all the disadvantages of axillary temperature, increased. It is very inaccurate. It requires flexing the thigh over the abdomen and keeping it in that position while taking it.

Temperatures differ depending on the measurement site. For example, rectal temperatures are typically 0.4°C (0.7°F) higher than oral temperatures, while axillary temperatures are 0.6°C (1°F) lower than oral temperatures.

The oral temperature of a healthy person is 37 °C.

The inguinal or axillary temperature of a healthy person is 0.4-0.6 °C lower; i.e. around 36.5°C.

The rectal or vaginal temperature of a healthy person is 0.4-0.5 °C higher than the oral temperature; i.e. around 37.5°C.

The axillary temperature should not exceed 37 °C, the oral temperature being up to 37.3 °C and the rectal temperature up to 37.5 °C.

It should be noted that in a certain number of healthy subjects the axillary temperature exceeds 37°C.

This "thermal floridity" has been reported by Czpai (among 22,000 subjects examined) in 45% of men and 54% of women.

Physiologically, the temperature experiences variations of up to 0.8 °C during the 24 hours of the day, with a maximum between 5 and 8:00 p.m. and a minimum, around 4:00 a.m. People who work at night have this daily variation reversed. Food and muscular exercise increase temperature, proportional to the intensity of the exercise performed.

The temperature should always be taken on an empty stomach, before lunch, in the afternoon, before lunch, at night and in the early morning.

The temperature should not be taken during the digestive period, as this will cause it to rise due to digestion, nor should it be taken in the mouth when a hot or cold drink has just been consumed; nor in

the armpit or skin surface, when hot or cold bags have been kept in them. Administration of oxygen with a mask may also affect the reliability of the oral reading, because inhaled oxygen has a high water content that can cool the oral mucosa.

When there is a febrile process that requires it, the temperature should be taken every 4 hours.

For a reliable measurement, the thermometer must be inserted properly and left in place for the required time. Numerous studies estimate that the optimal time to keep glass thermometers in place is 8 min.

Electronic thermometer readings require much less time, often only 10 s. Rectal glass thermometers can be left in place for 3 min.

Guide to axillary body temperature measurement

1. Prepare the equipment:

a) Mercury (Hg) or glass thermometer. If the thermometer is stored in a disinfectant solution, wipe it with a cloth, or rinse it with cold water. Dry it from the end of the bulb to the end of the blunt end, rotating the fabric to cover the entire surface.

If the reading on the thermometer is greater than 35°C, 95°F, shake it down. To do this, hold the thermometer with your index and middle fingers and shake it by swinging your wrist.

Be careful not to break the thermometer by shaking it too close to a hard surface. (bureau, table, stretcher).

b) Electronic thermometer. Disconnect it from the battery charging unit. Cover the metal tester with a disposable plastic cover and place it firmly inside the cover until it falls into place.

2. Insert the thermometer:

a) Expose the armpit and place the tip of the thermometer in the armpit. Fold your arm across your chest and hold it in place.

The large blood vessels in the area reflect internal body heat.

b) Keep the glass thermometer in place for 8-10 min and the electronic one, according to the manufacturer's instructions, usually until you hear a signal.

Although the optimal holding time of the glass thermometer is 8 min, the peak temperature is often recorded after 4-5 min.

3. Read the thermometer:

a) Glass thermometer. Remove the thermometer and wipe away any discharge with a cloth, working from the end toward the bulb. Hold the thermometer at eye level and read at the bottom of the mercury column.

b) Electronic thermometer. Remove the thermometer and observe the digital reading displayed.

4. Store the thermometer:

a) Glass thermometer. Wash it with soapy water; then, dry it and return it to the disinfectant substance or protective case, after lowering it.

b) Electronic thermometer. Remove the disposable plastic cover and return the thermometer unit to the battery charger.

Guide to rectal measurement of body temperature

1. Prepare the equipment:

a) Use a rectal, glass or electronic thermometer. Prepare the rectal thermometer using the same procedure as for the oral thermometer.

If the thermometer is electronic, remember to insert a disposable cover to keep it aseptic.

b) Lubricate the thermometer with water-soluble petroleum jelly before inserting it.

2. Insert the thermometer:

a) Place the patient on his side, with his knees slightly bent. Expose the anus by separating the buttocks.

b) Insert the thermometer 1.5-4 cm. Never force insertion, as this may cause injury or puncture. Also keep in mind not to insert it into feces.

c) Keep the glass thermometer for 3 min and the electronic one, according to the manufacturer's instructions.

3. Complete the procedure:

Dry the thermometer and follow the usual procedure for reading axillary temperature.

Guide for oral measurement of body temperature

1. Prepare the equipment:

a) Use a glass or electronic thermometer and follow the same procedure for preparing the equipment as you would for the thermometer in the axillary intake.

Before inserting the glass thermometer, unpleasant-tasting solutions must be removed. Clean from "I clean the dirty" to maintain asepsis. The bulb must be very clean, because it will go inside the mouth.

2. Place the thermometer:

a) Ask the examinee to open their mouth and place the tip of the thermometer in the right or left sublingual pocket (on either side of the frenulum) and instruct them to hold it under the tongue.
b) Keep the glass thermometer in place for 5-10 min and the electronic one, according to the manufacturer's instructions.

3. Complete the procedure:

Dry the thermometer and follow the usual procedure for the thermometer used in axillary feedings.

SKIN, MUCOUS AND FANERA (HAIR AND NAILS)

General guidelines

Examine the skin, mucous membranes, hair and nails using inspection and palpation as basic techniques.

You should also be equipped with a measuring tape or ruler to measure skin lesions, and gloves for palpation if it is necessary to prevent contact with body fluids. Inspect the skin and hair meticulously, thoroughly, from head to toe.

The general inspection of the integuments, as for the inspection of any other aspect, must be carried out in a place with adequate temperature (20-25 °C) and with sufficient diffuse lighting, natural or artificial of a natural type, that allows faithful evaluation of the colors and ensures an effective and meticulous examination. In certain cases, examination in incident or tangent light is also useful, because certain details become more apparent.

Rooms with a temperature lower than that indicated give rise to the appearance of paleness, tingling, tremors, etc.; and those with a higher temperature, redness, sweating, venous dilation, etc.

tera.

Premises illuminated with artificial light that is not natural (incandescent light bulb, candles, etc.) determine the appearance of non-existent colors (yellow, livid, reddish, depending on the light) or can mask those that may really exist.

All these facts, depending on the inadequate conditions in which the inspection is carried out, can lead us to errors; hence the warning to avoid all carelessness and to take into account even the smallest details.

The integuments must be examined in their entire extension, discovering and observing, part by part, the body surface; always trying to take the necessary care and tact so as not to hurt the modesty and delicacy of the subject, because modesty and clumsy maneuvers alter the physical conditions of the skin and psychologically predispose the examinee against the doctor or the examiner.

The integumentary inspection is complemented by the examination of the oral mucosa and ocular conjunctivae.

If abnormalities are detected, always compare the right and left sides of the body. For example, if your left foot appears cold and sweaty, examine your right foot for similar signs. Is there an abnormal appearance in a nail bed? In all 1nail beds? A rash appears on one arm and not the other? Make a further evaluation of the detected abnormalities by interviewing the person and asking how long they have been present; if there is associated discomfort or pain; what exacerbates or improves the findings; and what other injury or illness may be involved.

FUR

For a thorough examination of the skin we must undress the patient, respecting, however, the mandates of modesty, as we said previously, and avoiding iatrogenesis, especially if it is a woman, in which we will only do it partially and with the greatest delicacy. If there is no examination or examination gown (gown above the knees, sleeveless, closed at the front and open at the back, with strips of fabric to fasten at the neck, back and waist), the person can remain in their underwear and cover themselves with a sheet, towel or appropriate fabric, which must be available.

wearable. If the premises do not offer the necessary privacy, a paraban must be placed. If the person being examined is young and of the opposite sex, it is advisable to be accompanied by other health personnel, such as a nurse, or a family member, to avoid embarrassing situations and misunderstandings.

If the subject walks or the examination is performed in an outpatient clinic, the skin of the exposed parts (extremities, head, neck, back and male chest) is generally examined first with the person sitting, and later the rest, uncovering the area to be examined in parts, with the person lying down. If the subject is bedridden and the examination is performed in the hospital or at home, the skin examination can be performed completely, with the person lying down.

Only normal findings will be detailed in this Section. The abnormalities will be studied in Clinical Propedeutics.

Guide to skin examination

The aspects that should be explored are:

1. Through inspection:
   a) Color and pigmentation.
   b) Hygiene and injuries.
2. Through palpation:
   a) Humidity.
   b) Temperature.
   c) Texture and thickness.
   d) Turgor and mobility.

Skin inspection

Color and pigmentation

Normal skin color varies according to different races, ages and regions of the body. Seasonal and occupational variations will also be taken into account.

Well known is the division that anthropologists, based on the color of the external integument, have made of the different human races, into white, yellow and black.

We do not ignore how the color of the skin varies at different ages, going in the white race, for example, from the pinkish white of the first months of life, to the white of the adolescent and adult, and to the yellowish white or dark white of the most advanced ages.

Likewise, there are normal variations in the skin color of certain regions of the body, particularly lar on the face, extremities, genitals and parts exposed to friction or the elements (waist, forearms, etc.).

We know that certain regions of the body have a darker color than others, such as the genital organs, the abdominal line, the nipples, the areola and the skin that surrounds the natural orifices. We also know that the skin regions that are ordinarily exposed, exposed to the action of the sun's rays, are darker than those that usually remain protected by clothing.

Ethnic variations in skin and mucous membrane color

Light pigmented breed. From ivory to pink, with possible olive or yellow overtones. Exposed areas are usually darker than unexposed areas.
In people with very light skin, it is common to find small hyperpigmentation known as freckles (ephelides), on the face, chest and arms.

Dark pigmented breed. Tan to dark brown in color. The lips may have a bluish tone in those of Mediterranean descent. Blacks may have a reddish or blue color to the lips and mucous membranes.

Yellow skin unrelated to ethnicity may be due to pigment retention or calluses, when confined to a specific area.

Hygiene and injuries

Hygiene. Hygienic practices associated with the skin vary greatly. Frequent bathing is determined physiologically, socially and culturally. Clean skin is usually healthy.

Injuries. Normal skin is generally free of lesions, but small excoriations, scars, folds, freckles (ephelides) and moles (nevus) may be observed.

During skin inspection, always expose vulnerable areas such as skin folds and pressure points. In the folds, observe those in the inguinal regions and the inframammary folds in pendulous breasts, where excess moisture can contribute to the growth of pathogenic microorganisms and skin erosions.

If the patient is bedridden or immobilized, focus your attention on the skin overlying the body's pressure points, where injuries may occur in the early stages of formation, such as pressure sores.
In addition to the bony prominences of the occipital, scapulae, sacrum, greater trochanters and heels, other pressure points should be examined, including those areas where the skin is in contact with tubes used for treatment, such as the nostrils (nasogastric tubes),

lips (endotracheal tube) and ears (oxygen connecting cannula). Also examine the skin where tapes and restraints sit.

Also uncover wounds to assess healing and note any excessive drainage. Keep it as a practice, if it is not contraindicated.

In the Clinical Propedeutics Section, primary, secondary and vascular lesions of the skin will be seen.

Skin palpation

Palpation of the skin should be superficial and light, or forming small folds between the thumb and index finger or other fingers of the hand; In this way the following qualities are studied.

Humidity

Normal skin is generally dry to the touch, but moisture can accumulate in skin folds. A slightly warm and moist sensation is felt if the person is in a warm environment, and during exercise or recovery to cool the body. Anxiety can cause clammy palms and sweating in the armpits, forehead, and scalp.

Dryness, profuse sweating (diaphoresis), or excess or deficiency of fat may be abnormal, but it does not always have clinical significance. Specify whether these conditions are generalized or localized to certain areas.

Temperature

Normothermic skin is warm to the touch. Cold skin temperatures can also be normal, if the coldness is accompanied by dry skin.

The decrease in temperature is observed in vasoconstriction, both physiological and during cold, as well as in pathological conditions, in which the skin is generally moist, sweaty.

Acute inflammation in any area of the skin is accompanied by an increase in local temperature.

Texture and thickness

Unexposed skin is smooth, while exposed skin may be rough.

The thickness of the skin varies; The epidermis covering the eyelids and ears can be 1/20 cm thick, while the epidermis on the soles of the feet can be as thick as 1/2 cm.

Keep in mind that very thin skin can be very friable and its integrity is easily broken, and that very rough skin can be normal.

Turgority and mobility

Normal skin has an elastic turgor and quickly returns to its original shape when pressed between the thumb and index finger. When there is cellular dehydration, the skin has poor turgor; It is slow to recover its original shape. Skin mobility is restricted in edema and scleroderma.

Skin examination record

Describe the characteristics of any skin examination (color and pigmentation, hygiene and lesions, humidity, temperature, texture and thickness, turgor and mobility). If there are skin lesions, although it cannot be determined whether they have pathological significance, the following must be described: number, location, shape, size, color, consistency, mobility and sensitivity.

Normal skin registration example

Fur. Pinkish white in color (normocolored), warm (normothermic), dry, without prolonged skin folding when pinched (normohydric), smooth, with normal turgor, elasticity, thickness and mobility, with good hygiene and without

FANERA (HAIR AND NAILS)

The fanera is represented by integumentary productions that are also known as skin attachments: hairs and nails.

The state of the pharynx, particularly the hair, has great semiological importance since, under normal conditions, it reflects, to a large extent, the way (in intensity and rhythm) in which the various stages of development are fulfilled, which is governed by the functioning of the endocrine system. In a pathological state, its alterations are symptoms that reveal the repercussions that morbid processes have on the neuroendocrine system and on trophism in general.

Hairs

Hairs are filiform productions of the epidermis that are not similar in terms of quality and other characteristics, but vary according to the part of the organism they cover and, therefore, receive different names: hair, those of the scalp; eyebrows, those of the supraorbital arches; eyelashes, those on the edges of the eyelids; beard, those on the face; mustache, those on the upper lip; hairs - properly speaking - those of the armpits, pubes and other regions of the body (particularly in men); vellus, the short, fine and light hair of the face and some regions of the body of the woman and the child: and lanugo or fluff, the rudimentary, very fine, very short and light hair that the infant has on the face and body.

hair examination

It is explored through inspection and palpation, trying to establish variations, both in state

normal or pathological, which may present its characteristics, which are:

1. Quantity.
2. Distribution.
3. Implementation.
4. Quality, which translates into: appearance, color, length, thickness, resistance and trophic state in general.

The characteristics that we have just enumerated suffer physiological variations, of an individual and general order, which it is necessary to know, because they reflect the normal state of many biological conditions, such as heredity, development and the balanced functioning of the neuroendocrine system. These conditions are represented by: age and sex, constitutional type and race.

Age and sex

The hairs constitute morphological characters specific to each period of life and each sex. Its characteristics reveal to us the way in which the development of the individual has been completed, whether it has been carried out normally or whether some constitutional or morbid factor has interfered and modified it. In this lies the value of your knowledge.

The characteristics of hair in the different stages of life are the following:

A. In infancy and childhood.

In these stages, hair development is constituted by primary trichogenesis, represented at birth by scarce hair, which is very fine and sparse;
for the eyebrows, which are rudimentary and fine; for the eyelashes, and for the lanugo or fluff, which is found on the face, on the back and on the outside of the upper limbs.

As the infant grows, the hair increases in quantity, as does the eyebrows, although they remain very thin, while the lanugo of the face is largely lost and that of the rest of the body, especially the limbs, tends to take on hair characteristics.

B. At puberty.

At this stage, the so-called secondary or terminal trichogenesis begins, characterized by the appearance of hairs in regions previously devoid of them and because the quality and quantity of the hairs that already existed change fundamentally. This secondary trichogenesis begins with the appearance of sparse hairs on the pubis during the first semester of puberty, that is, at twelve and a half years in girls and at thirteen and a half in boys, as has been demonstrated by Professor Dr. José R. Jordán, in Cuba, in his social research.

b growth and development. In the second semester, these pubic hairs increase in number and size, at the same time that in the male the change in the
VOICE.

In the third and fourth semesters, hairs appear in the armpits and the upper lip on the upper lip in males. In the fifth semester, in the girl, the pubic and armpit hairs have already acquired definitive characteristics, in their quality and quantity, and in the boy, trichogenesis has been completed with the appearance of a mustache and beard, so that in the five semesters that lasts - according to research by Paul Godin - the process of puberty (puber:
hairs; pubere: to be covered with hair) hairs have appeared in the regions that will be definitively covered by them, and at the same time, the lanugo or fluff of childhood has been replaced by hairs in men and by hairs in women.

C. In adulthood.

At this stage, the quantity, distribution and quality of the hairs configure a secondary sexual character specific to each sex, a character that, as we have seen, appears at puberty to be complete upon entering adulthood, in the following way:

In men, a generic characteristic of hair is that of being abundant and strong. The hairs are abundant, strong and set on the borders of the forehead, leaving two entrances in the superolateral corners of the forehead which, with age, deepen somewhat in the temporal region, and which R.O. Stein considers a characteristic of sex. On the face, abundant and thick eyebrows and the presence of a beard and mustache constitute another characteristic of virility. On the body, the hairs are rather abundant and somewhat thick and are distributed on the shoulders, back, chest - especially around the nipples - and on the pubis, where they are abundant, long and continue with those of the infra-umbilical region, forming an angle that has its vertex at the navel, an arrangement that is typical of the male sex (mount of Apollo) and, finally, on the limbs, where they are particularly abundant on the forearms and legs.

In women, a generic characteristic of hair is that it is rather sparse and fine. The hair is, compared to that of man, fine, long and less abundant, and is arranged on the forehead forming a half-moon open downwards. This disposition has the value of being a characteristic of femininity. On the face, the eyebrows and eyelashes are thin, long and not very abundant; and on the cheeks and upper lip there is only a very fine and sparse hair.

On the body, hairs with infantile characteristics are found on the outer region of the thighs, particularly

lamentably in legs and forearms. In the pubis, the hairs are arranged horizontally on the suprapubic symphysis, leaving the infraumbilical region free of them, a characteristic that is typical of the sex (mons of Venus).

D. At the critical age.

Here the grayness that began discreetly in the hair in the last five years of maturity, intensifies and becomes present, progressively, in the beard and in the hair of the body, at the same time that baldness is insinuated with the accentuation of the frontotemporal receding lines or by its appearance around the crown of the head, this in regards to men. In women, in addition to the graying of the hair, hard hairs with characteristics of a male beard appear on the upper lip and chin, which means, according to G. Marañón, an expression of the evolution of sex towards masculinity and for Tandler and Mathes an asexual regression.

E. In old age.

The characteristics that we have just seen become accentuated as the years go by; In addition, the hair loses vitality, falls out easily, gray hair becomes generalized and baldness becomes increasingly extensive.

constitutional type

In humans, individuals with a hypergenital, hyperthyroid, hyperadrenal and hyperhypophyseal constitution have thick and abundant hair and the characteristics of the sex are more accentuated; It can be said that they present a slight hirsutism. On the other hand, in those with a hypogenital, hypothyroid, hypoadrenal and hypopituitary constitution the hairs are very fine and less abundant and it can be said that they present a slight hairlessness.

In women, those with a hyperadrenal and hyperhypophyseal constitution present trichogenesis with a slightly virile appearance, and are, it can be said, slightly
In those with a hyperovarian, hypothyroid, hypoadrenal and hypopituitary constitution, the character of trichogenesis is frankly feminine or discreetly infantile.

Race

Rather than racial variations, it is appropriate to speak of variations in trichogenesis in large ethnic groups.

Among Europeans, the Nordics are characterized by their light hair, rather fine and not very abundant:
while those from the center, and more particularly those from the Mediterranean basin, have dark, thick and abundant hair, sometimes even showing true hirsutism.

In African blacks, very short, black, curly hair predominates, and the body is rather hairless.

Among Asians, straight, dark hair is common, and a discreet hairlessness on the body and beard.

In the native American races, the hair is rather thick, dark and unruly, and the body and beard are poor in hair; In general, they present a discreet cleanliness.

Within our population, the characteristics of the black race and its crossing with the Spanish predominate; Different characteristics are observed depending on the hereditary predominance.

Pathological alterations will be studied in Section II, fundamentally when treating the semiology of the endocrine system.

Guide to hair examination

When examining the hair, the following characteristics must be systematically evaluated: color and pigmentation, quantity, texture, distribution and hygiene.

1. Colory pigmentation. Natural: black, blonde, brown, gray, gray (gray, white). Dyed hair, although it must be specified, has no pathological significance and can mask its alterations.

2. Distribution. According to sex.

3. Quantity. Increases after puberty. Decreases with age.

4. Texture and thickness. Curly, curly, straight. Thick or thin. Brittle hair may have clinical significance.

5. implantation. Normal hair generally has good implantation. Hair that falls out easily may have clinical significance.

6. Hygiene. Neglected hair with poor hygiene is more likely to be associated with skin diseases (bacterial and mite). It can also express the evolutionary status of a patient. For example: a man who wants to shave denotes improvement.

Example of hair registration

Hair. Good hygiene; of normal color and pigmentation, quantity, texture and distribution, according to age, sex and race.

Nails

The nails are represented by the horny plates on the back of the phalanges of the fingers, which are formed due to a special keratinization of their skin. They must be studied for their shape, appearance, resistance, growth and color, which can present alterations in the most diverse internal diseases, particularly in those of a metabolic, infectious, neurovegetative nature, etc.

The physiological variations of these ungular characteristics due to age, sex and constitutional type are well known, so we insist on them; We will only remember that the nails of individuals with an asthenic constitution (longilinear), particularly those with hyperthyroidism, are perfectly shaped, shiny, grow well (about 3 mm per month) and resistant; while in asthenic people, particularly in hypothyroid people, the nails are fragile, thin, slow growing and not very perfect in shape.

Guide to nail examination

Normal findings

1. Shape and configuration:

a) Dorsal surface slightly convex.

b) Thickness 0.3-0.65 mm.

c) Angle from the base of the nail to the skin-nail interface: 160°.

2. Color. Uniform, except the difference between the lunula and the rest. They appear pink in white individuals and can be bluish in blacks.

3. Capillary refill time. Less than 3 s.

Squeeze the nail between your thumb and index finger; When the pressure is released it will appear whitish. It is the period of time in which the nail bed regains its base color.

The normal nail grows about 0.5 mm per week; It is pink in color, with a smooth surface and elastic consistency. The whitish lunula occupies a fifth of its surface.

Deviation from normality

Platonychia. Flat nail.

Coiloniguia. Concave, spoon-shaped nail. Associated with iron deficiency.

Nail on watch glass. Angle greater than 160°. Associated with chronic tissue hypoxia.

Onychophagia. He bites his nails.

Onychorrhexis. Fragile, split nails with an irregular edge.

Onychomycosis. Irregular whitish lesions, produced by fungi.

Onichauxris. Nail hypertrophy.

Beau lines. Transverse cracks produced by malnutrition due to severe illness.

Example of nail registration

To record the nail examination, you must proceed in a similar way as you did when recording the hair, describing each of their characteristics.

Uias: shape and configuration, color, lesions.

Capillary refill time is generally not part of the daily physical examination in our country and is part of the examination of the cardiovascular system, but you should get used to exploring it at this time, to avoid regrettable forgetfulness.

The examination of the subcutaneous cellular tissue and its adipose tissue will be studied in Section II.

GENERAL PHYSICAL EXAMINATION RECORD MODEL

1. Biotype. Normolinear, brevilinear, longilinear.
2. Facies. Expressive, happy, anxious, angry, sad, in pain, etc.
3. Attitude or posture:
   a) Standing and sitting. Erect, correct.
   b) Lying down or in bed: Active, non-obligatory position (supine, prone, lateral).
4. Ambulation or walking (fluidity and coordination of movements) . Fluid and coordinated gait.
5. Weight.
6. Size.
7. Temperature.
8. Skin. Color and pigmentation, humidity and turgor, temperature, elasticity and thickness, mobility, hygiene and injuries.
   (If lesions describe quantity, location, color.
   type, shape, size, consistency, sensitivity, mobility).
9. Mucous membranes. Color and humidity (normocolored and normohydric). Pigmentation and lesions.
10. Hair. Color and pigmentation, distribution, quantity, texture and thickness, implantation and hygiene.
11. Nails. Shape and configuration, color, capillary refill time. Injuries.
12. Subcutaneous cellular tissue (SCT). No infiltration (visible bone prominences, no footprint or pressure godet).
13. Adipose panniculus. Preserved, increased, decreased.

Note: In secondary care medical records it is customary to include weight, height and temperature at the end of the general physical examination.

4 4

Once the general physical examination has been completed, we will do the physical examination by segments or regions of the body. These regions are:

-Head.

-Neck.

- Chest.

Backbone.

- Abdomen.

Extremities.

As most of these regions will be addressed in the study of the exploration of the different systems, we will limit ourselves here to pointing out the most important aspects that must be explored in each of these segments, and the exploratory techniques that will not be addressed later. We remember that we will use the four classic methods described in the physical examination, that is, inspection, palpation, percussion and auscultation.

HEAD EXPLORATION

The head includes the skull and the face.

SKULL

First, the position, shape and proportions of the head as a whole are explored by inspection, and its height by measurement or appreciation. Only when certain pathological situations are suspected, the skull is percussed and auscultated.

The shape and proportions of the head as a whole vary according to constitutional type, race, age and sex. Thus, in the breviline the transverse head diameter (brachycephalic) and the facial procidences linked to the masticatory apparatus (lower jaw and masseter) predominate; in the longilinear the sagittal diameter is greater (dolichocephalic), and the elements linked to the respiratory system (nose, malar, supraorbital arches) are accentuated; In the normolinear the proportions are usually harmonic.

The height of the head, from the vertex to the chin, is 18-20 cm, that is, 13% of the body length; It is proportionally greater in women and newborns than in men.

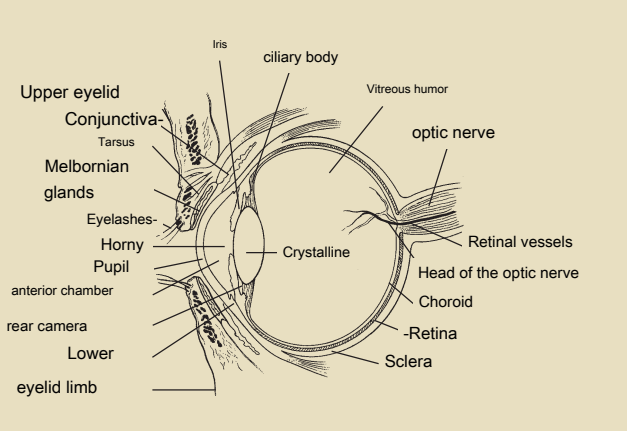


Fig. 4.1 Longitudinal section of the eye.

Additionally, inspect and palpate the skull, evaluating its symmetry, the absence of pain or irregularities of the scalp and the type, qualities and implantation of the hair. Take advantage now and perform palpation and auscultation of the temporal arteries, above the temples, whose exploration will be detailed in the chapter on the peripheral vascular system.

FACE

In its exploration we will take into account the following aspects:

1. Inspection and palpation of the face.
2. Exploration of the external structures of the eye.
3. Examination of the nose and perinasal sinuses.
4. Exploration of the external and internal structures of the mouth.
5. Examination of the external ear.
6. Exploration of the cranial nerves.

Inspection and palpation of the face

In the face we will explore by inspection: its shape, the trophism and the symmetry of the furrows of the forehead, the cheeks and the lips, both at rest and when spontaneous movement when speaking or when ordered, and the shape and symmetry of the jaw and chin. Palpation rules out the existence of pain or tumors.

It is advisable to explore the perinasal sinuses by palpating painful points when the sinuses are inflamed (sinusitis), as we will see later.

Exploration of the external structures of the eye

Brief anatomical account

The visible structures of the eye include the conjunctiva, sclera, cornea, iris, pupil, and ductal openings of the lacrimal sac.

The conjunctiva is a membranous tissue that covers the inner portion of the eyelids (palpebral conjunctiva) and the sclera of the eyeball (bulbar conjunctiva).

The sclera is a hard, fibrous layer that surrounds the eyeball, except in its most anterior portion.

The limbus marks the point of union of the sclera with the cornea, the first transparent structure that allows light to enter the eye.

The pigmented iris surrounds the pupil.

The pupil, the central opening of the iris, changes size according to the stimulation of the iris sphincter and the pupillary dilator muscles, as well as the ciliary muscle, located behind the sclera (fig. 4.1).

These muscles, called intrinsic muscles of the eye, are innervated by cranial nerve III (oculomotor) and by nerve fibers coming from the ciliary ganglion.

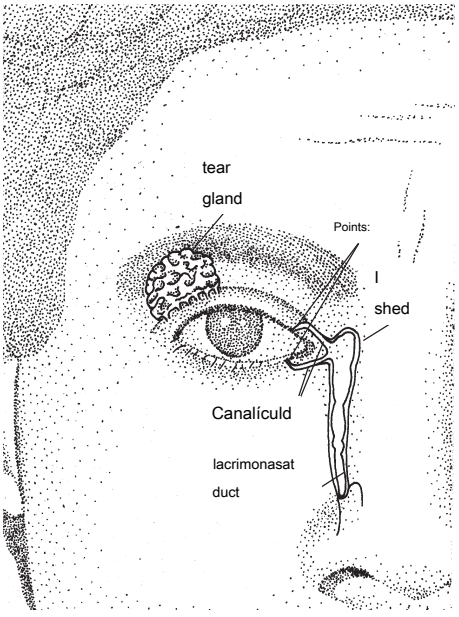
Pupil size is determined by the balance between sympathetic and parasympathetic discharges.

The lacrinal apparatus, as shown in Figure 4.2, includes the lacrimnal gland, which produces tears to lubricate the eye; the puncta, a hole in each of the eyelid edges, located on the sides of the inner canthus of the eye, which drains tears into the lacrimal canaliculi, towards the lacrimal sac. The tears are finally drained towards the nose, through the lacrimonasal duct.

Guidelines for examination and recording of data

A complete eye examination, excluding vision screening, includes examining and recording the following:

Fig. 4.2 Lacrimal apparatus.



- Lacrimal apparatus: lacrimal gland and sac, puncta and tears.

-Conjunctiva: palpebral and bulbar.

- Sclera, cornea, iris and pupils: shape, size, color.

- Pupillary reflexes, retinal light reflex.

- Retina: color and pigmentation, vessels, macula, optic disc.

- Eye movements: conjugate gaze, movements of the extrinsic ocular muscles.

- Intraocular pressure.

In the regional eye examination we will examine the conjunctivae, sclera, cornea, iris, pupil and lacrimal apparatus. Furthermore, if there is synchronism of eye movements upon simple inspection, and if there is protrusion of the eyeballs (exophthalmos) or the opposite (enophthalmos).

The examination of the external structures of the eyes also includes the examination of the eyebrows, eyelids and eyelashes.

The rest of the aspects mentioned, along with the vision examination, are explained in detail, when studying the examination of the nervous system, in this same Section.

Intraocular pressure is measured by specialized personnel and is not the objective of this book.

Techniques for examining the external structures of the eyes

Stand facing the subject, who should be seated on the examination table, so that both eyes are at a similar level. It can also be done with the explorer and the explored person, sitting face to face. The examination standing is not performed, even if both are of similar height, because it is uncomfortable for both.

1. Inspect the eyebrows, eyelids and eyelashes, and note the shape of the eyes and their symmetry.
2. Examine spontaneous blinking for several seconds to detect complete closure of the eyelids. Specify the position of the eyeball and whether there is any other abnormal eye or eyelid movement.

Normal findings

Symmetry of the eyes and associated structures. The shape of the eyes varies from round to almond-shaped. In Asians, the fold of skin over the inner canthus (epicanthus) causes the shape of the eye to appear narrower.
The eyes may appear larger and rounder in some black people because the eyeball protrudes slightly in front of the supraorbital rim.

Eyebrows. Variable appearance according to genetic background. Women tend to "fix" their eyebrows, with partial or total extraction or plucking of them, which makes it difficult to detect abnormalities.
In unmodified eyebrows, there should be no absence of hairs (alopecia) in any of their parts.

Eyelashes. Curves outward, away from the eyes.
Not alopecia.

Eyelids. The upper eyelid does not cover the pupil when it opens, but may cover the upper portion of the iris; The eyelids should open and close completely, without drooping or delaying one of them, or both.

3. Inspect the internal conjunctiva and sclera.

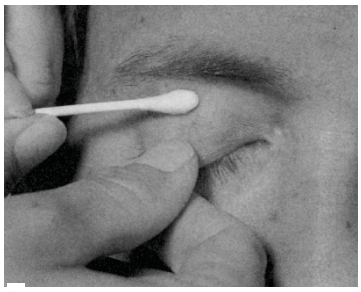
Kindly stretch the lower eyelid down with your thumb, to see the lower palpebral conjunctiva. Note the appearance of the exposed sclera (Fig. 4.3). To adequately see the upper palpebral conjunctiva and the sclera covered by said eyelid, place your hand opposite the eye being examined, on the subject's forehead, so that the thumb is down. Pull the eyelid up, gently with your thumb, and ask the person to look down and in. Observe the color of the sclera carefully, especially in its superolateral portion, since it is the place where slight abnormal changes in its color are best detected, such as the light yellow tint of sub-jaundice.

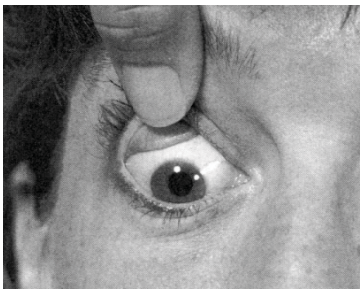
These structures can also be inspected, optionally, by careful eversion of the upper eyelid (Fig. 4.4). To do this, you need to have an applicator, with or without cotton on the tip. Ask the subject to look down with his eyes slightly open. Kindly grab the eyelid between your thumb and index finger at the eyelid/eyelash junction and pull down slightly. With your free hand, place the applicator horizontally on the lower portion of the eyelid, without pressing it against the eyeball, while pulling the eyelid upward to evert it.

Remove the applicator and hold the eyelid against the superior bony orbit to complete the inspection.

Fig. 4.3 Inspection of the lower palpebral conjunctiva.

Fig. 4.4 Eversion of the upper eyelid: a, step 1; b, step 2.





Inspect the upper palpebral conjunctiva, the appearance of the exposed sclera, and also note the small visible portion of the lacrinmal gland. Then, pull the eyelid forward slightly. It will return to the normal position as you release it and the subject blinks. Repeat this procedure on the other eye.

We reiterate the importance of observing the color of the ocular conjunctivae and sclera, places where the explorer usually prioritizes the search for anemia or accumulation of abnormal pigments.

Normal findings

Palpebral conjunctiva: pink, moist and without lesions.

Bulbar conjunctiva: transparent, allowing the white sclera to be seen through it.

The small conjunctival blood vessels, which are normally not dilated, can be visualized.

Normal sclera: white, pale yellow tinge in some black subjects.

4. Inspect the rest of the visible ocular structures.

Use a flashlight to tangentially illuminate the lens and cornea. Inspect from various angles, noting surface characteristics and absence of opacities. Observe and compare the shape of the iris and its color, and the shape and size of the pupil.

Normal findings

Coneal: smooth, clear, transparent, with convex curvature.

Iris: color varies (blue, brown, gray, green with markings). The shape is round.

5. Examine the lacrimal apparatus by inspection and palpation (optional).

Gently stretch the lower eyelid with your thumb to expose the holes just lateral to the inner canthus of the eye (the puncta) and use a manual light source (pocket flashlight, ophthalmoscope) to better analyze their visualization.

Gently press the lacrimonasal duct with your index finger, just inside the lower orbital rim (Fig. 4.5), especially if you suspect blockage of the duct, indicated by excessive tearing (epiphora). Observe if any secretion comes out of the puncta. Proceed carefully, because the area may be tender if it is inflamed. Repeat the procedure on the other eye.

Normal findings

The puncta visible, but without excessive discharge unless the person is crying or the area is momentarily swollen. Lacrimal sac and gland not palpable or painful; moist eye surface.

Examination of the nose and perinasal sinuses

Examination of the nose and perinasal sinuses should be performed during the regional physical examination of the head or when the patient reports problems with these areas.

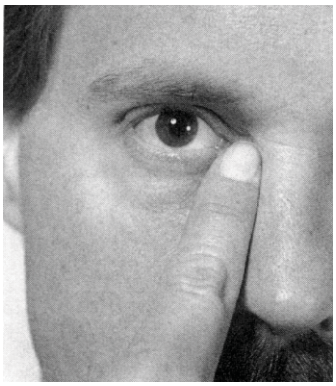
The most common problems associated with the nose are obstruction and irritation or hypersecretion, secondary to colds or allergies, and trauma.

The primary symptom of sinus problems is pain that occurs during inflammatory processes. Blockage and dental problems are the most common causes of sinus inflammation.

Anatomophysiological count

The primary functions of the nose include olfaction and the warming, humidification, and filtration of inspired air. Olfaction is evaluated by exploring the cranial nerve I (olfactory) (see Chapter 15 in this Section).

Fig. 4.5 Palpation of the lacrimal apparatus.



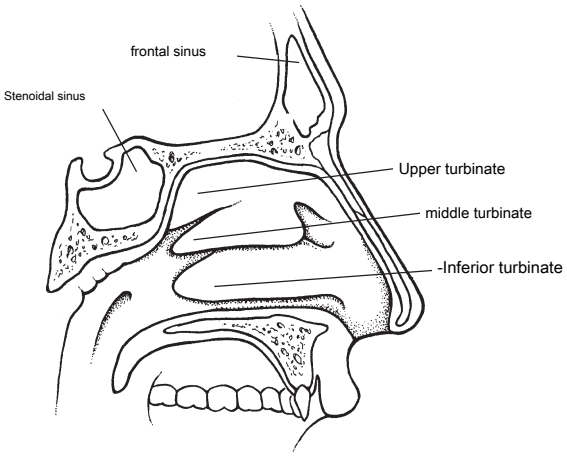


Fig. 4.6 Nasal turbinates.

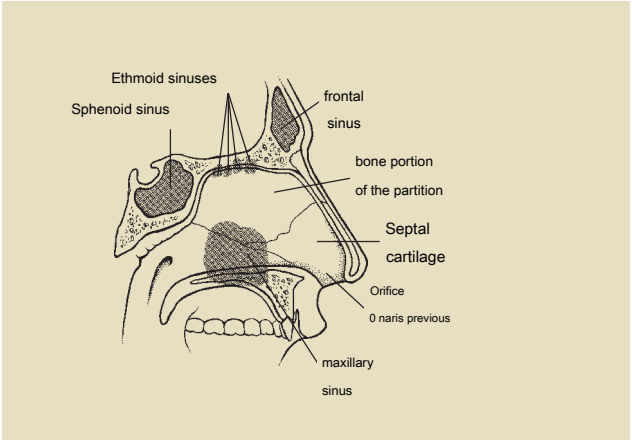


Fig. 4.7 The perinasal sinuses.

The inspired air is heated and humidified as it passes through the turbinates. The turbinates consist of three bony projections from the lateral walls of each nasal cavity, which are lined with ciliated epithelial cells, a large vascular bed, and mucus-secreting cells (Fig. 4.6).

During the physical examination, only the inferior and middle turbinates are visible.

The sinuses drain into the nose through small holes in the turbinates. When the turbinates are edematous, as can occur in the common cold, these orifices can become blocked. As a result, secretions can be stored in the sinuses and provide a medium for bacterial growth. The pressure of the accumulated fluid contributes to the pain of acute sinusitis.

The perinasal sinuses (Fig. 4.7) are the sphenoid sinuses, the ethmoid sinuses, the frontal sinuses, and the maxillary sinuses. Only the frontal and maxillary sinuses are accessible to physical examination. Normally the sinuses are filled with air and have no known function in humans. They are lined with mucus-secreting cells and drain into the nose.

Exploratory techniques

The most important aspects to take into account for the nose and sinus examination and its written record are:

Nose: shape and configuration, position and integrity of the nasal septum, patency of the nostrils, color of the mucous membranes, color and volume of the turbinates, secretions, lesions, masses and foreign bodies.

Sinuses: pain or not on palpation of the frontal and maxillary sinuses and quality of transillumination, if it is performed.

Nose exam

Examine the nose by inspection to evaluate its appearance, shape, size and rule out injuries and deformities. The patency of each nostril should be closing one side of the nose and then the other with digital pressure, and asking the person in each maneuver to "breathe out" through the nose.

The external shape of the nose varies greatly due to genetic differences and alterations secondary to trauma or reconstructive surgery. Deviations in the shape and configuration of the nose are generally not significant, unless they are an expression of recent trauma or are associated with airway obstruction. Areas of recent increase in volume should be palpated for inflammation.

The anterior or external nostrils must be patent. Masses or foreign bodies can interfere with airway patency (fig. 4.8).

The internal nose is inspected by tilting the head back and looking through the external nostril to visualize the vestibule, septum, and inferior and middle turbinates.

To allow visualization of the internal structures, place your thumb on the tip of the nose to move it upward and with the other hand hold a light source (flashlight, ophthalmoscope, otoscope) to illuminate the internal structures.

Inspection of internal structures can be facilitated by the use of a nasal speculum, but it is rarely used in lay physical examination.

Fig. 4.8 The nose.

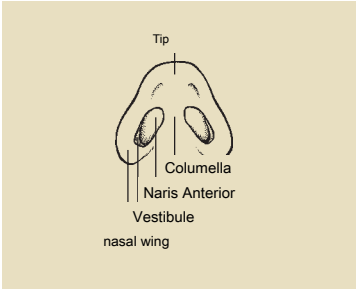
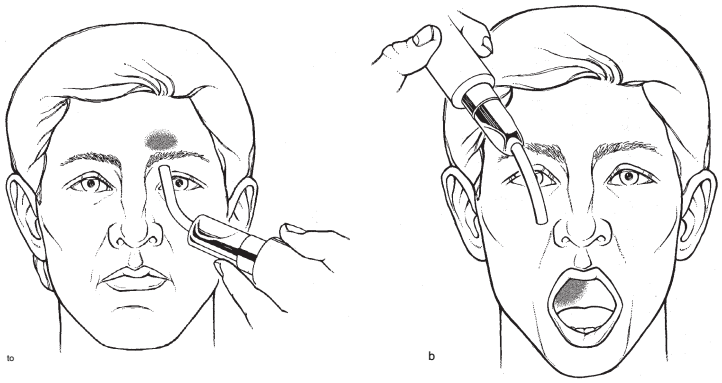


Fig. 4.9 Transillumination of the breasts: a, left frontal; b, right maxilla.



When using the speculum, take special care to avoid contact with the sensitive nasal septum.

Normal nasal septum: it should be straight, but its deviation is common. Severe deviation can interfere with patency. The partition must not be perforated.

Normal mucous membranes: their color is pink or matte red. A small amount of clear watery discharge may be seen.

Perinasal sinus examination

The frontal and maxillary sinuses are examined by palpation to detect pain related to inflammation.

Palpate the frontal sinuses by pressing your thumbs over the inner portion of the eyebrows. Avoid putting incorrect pressure on the eye sockets.

Palpate the maxillary sinuses by pressing the tips of your thumbs on the maxillary bones, located below the orbits and on the sides of the nose.

If pain is evident on palpation of the sinuses, transillumination can be performed to detect that they are occupied by liquids or masses.

Transillumination

Proper breast transillumination technique requires complete darkening of the room and an intense light source. The light of the otoscope can be used.
However, even with good technique, the examination findings may not be reliable, due to the great variability that exists in the transillumination observed among individuals with normal, air-filled breasts.

Transilluminate the frontal sinuses by firmly pressing the light source against the medial supraorbital arch.
Obvious redness over the eye socket is considered normal. If redness is not observed, the sinus must be occupied (fig. 4.9 a).

Transilluminate the maxillary sinuses by asking the subject to tilt his head back and open his mouth. Press the light against the skin, just below the inner rim of the orbit. Redness should be observed in the area of the hard palate (fig. 4.9 b).

Examination of the external ear

Anatomophysiological count

The external ear includes the pinna (ear) and the external auditory canal.

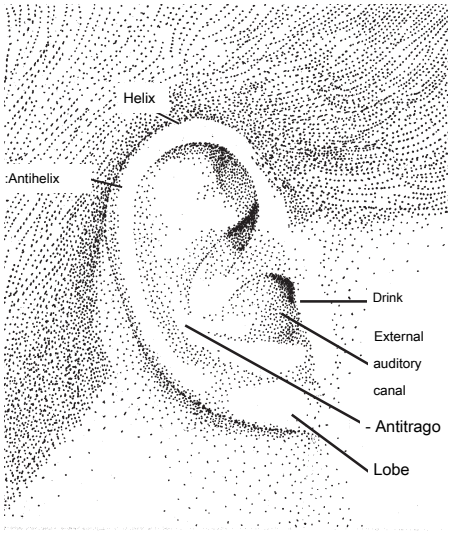
The ear is slightly curved, to receive sound waves, and its structures include: the helix, auricular tubercle, antihelix, antitragus, concha, tragus and lobe (fig. 4.10).

The ear canal is about an inch long, curves slightly, and ends at the tympanic membrane. The ear canal is supplied with many nerve endings and is extremely sensitive to touch, an important fact to remember during the examination.

Cerumen (ear oil) is secreted into the ear canal and can accumulate in the form of a hardened, brown-black plug.

The tympanic membrane, or auditory resonator, is a fibrous, movable tissue that separates the outer ear from the middle ear (fig. 4.11). Sound waves entering through the external auditory canal cause the tympanic membrane to vibrate, and the sound frequencies are transformed.

Fig. 4.10 The pinna.



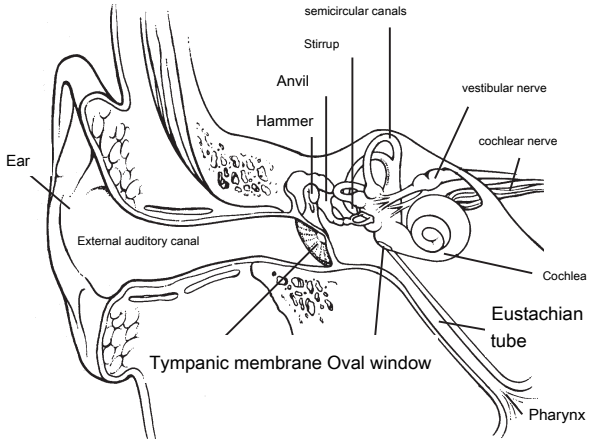


Fig. 4.11 Middle and inner ear.

They control, in turn, the small bones of hearing in the middle ear.

General guidelines

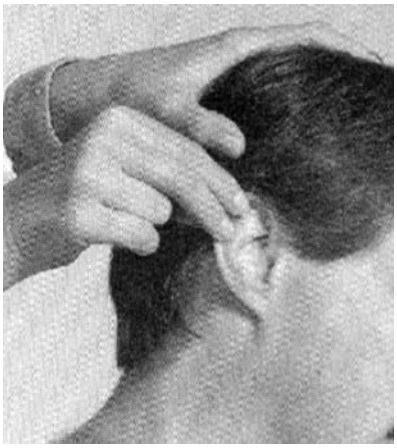
The ears are examined by inspection and palpation.

The otoscope is used to illuminate and inspect the ear canal and tympanic membrane.

Most otoscopes have a handle that contains the batteries, a switch to turn on the light, a viewing window, and the examination tip that can accommodate specula of different sizes (disposable or non-disposable). Choose the largest speculum that can be inserted into the ear without causing pain. Reusable specula should be carefully disinfected.

A pneumatic otoscope is used to determine the mobility of the tympanic membrane. A rubber bulb is attached to the otoscope and the examiner squeezes it while viewing the tympanic membrane. The squeezing action injects air, which normally causes the tympanic membrane to move in and out. The loss

Fig. 4.12 Palpation by pulling the ear up and back looking for pain.



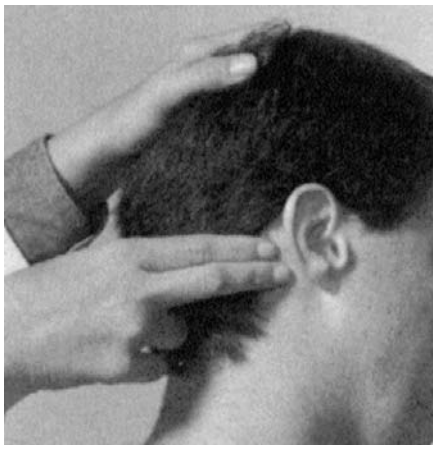


Fig. 4.13 Mastoid palpation for pain.

eardrum mobility is associated with middle ear infection.

Guidelines for examination and written recording of data

Pinna (ear): shape, size, symmetry, position, integrity of the skin, response to palpation of the tragus and the mastoid region, the mastoid bone.

External auditory orifice and canal: permeability, secretion, inflammation, hair growth, earwax.

Tympanic membrane: color, surface characteristics, limits, light reflection, configuration.

Exploratory techniques

A. Examine the external structures of the ear.

1. Inspect the integrity of the skin, the shape and size, the symmetry and position of the ears.
2. Palpate the ear between the thumb and index finger and check for pain or injury (fig. 4.12).
3. Palpate the mastoid region, which should not be painful.

The pain is associated with inflammation of the middle ear (fig. 4.13).

4. Press the tragus inward toward the ear canal to detect pain, which may indicate inflammation of the ear (fig. 4. 14).

Normal findings

The right and left ears are of the same size and shape. There should be no skin lesions.

Position of the ear: its upper margin should just touch or slightly cross an imaginary line drawn from the outer canthus of the eye to the occiput.

Lower set ears are associated with congenital kidney disorders and other chromosomal abnormalities.

B. Examine the ear canal with the otoscope.

Fig. 4.14 Palpation of the tragus.

Note: If the subject has symptoms of an ear infection (pain), examine the unaffected ear first. All measures should be taken to avoid transferring infected material from one ear to the other in the speculum.

1. Briefly explain the procedure. Ask the subject to direct his head slightly toward the opposite shoulder, away from the side being examined. Align the ear canal with the examination instrument.
2. Choose the largest speculum that fits comfortably in your ear and turn on the otoscope light.
3. Grab the otoscope with your dominant hand and hold it in one of two ways:

   For restless children and adults, place the hand holding the otoscope against the person's head to help stabilize the instrument (Fig. 4.15 a).

   For cooperative people, you can hold the handle so that it is in the correct position;
   stabilization efforts are unnecessary (fig. 4.15 b).

Fig. 4.15 Insertion of the otoscope: a, in children and restless adults; b, in calm adults.

4. With your free hand, grab the upper portion of the ear and gently pull it up, out and back, if the patient is an adult (see fig. 4.12). The ear is pulled down in infants and young children.
   This maneuver will straighten the duct so that you can see the tympanic membrane.
5. Insert the otoscope and advance gently to examine the surface of the external auditory canal through the lenses.

Normal findings

The adult ear canal is one inch long. The skin is intact, without redness or secretions. The channel is clean, without obstructions.

Hair growth is variable; hair grows near the outer third of the canal.

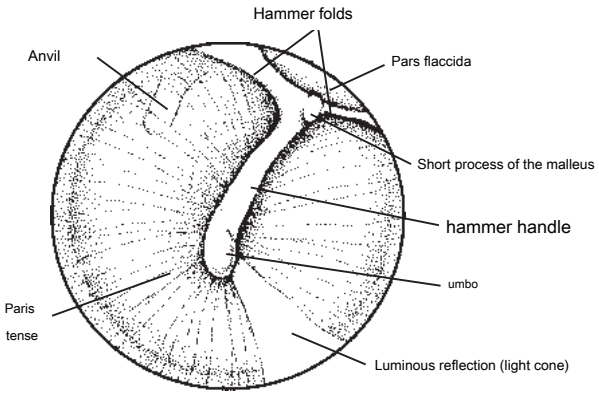
The color and consistency of earwax varies, depending on the length of time the secretion lasts. Fresh earwax is light, dark, or pinkish yellow, and is soft. Old earwax is light or dark carnmelite, and is hard.

C. Examine the tympanic membrane.

1. Next, move the otoscope to visualize the tympanic membrane. If this membrane is not initially visible, pull the ear up and back again, for further rectification of the ear canal. Do not force the speculum too distally in one direction; There may be earwax in the canal, partially obstructing the view of the tympanic membrane. Again, gently realign the canal by moving your ear and try to look around the sides for the earwax particles.

2. Inspect the tympanic membrane and note the major demarcations and color (Fig. 4.16).
   Quadrants of the tympanic membrane: anterosuperior, posterosuperior, anteroinferior, and posteroinferior.

Fig. 4.16 Right tympanic membrane.



Normal color: pearl gray, shiny. The diffuse white plaques on the membrane are scar tissue from previous inflammations.

Surface: continuous and intact, slightly transparent

rent.

Demarcations: white light reflection (light cone) projected onto the anterior-inferior quadrant. The light reflection is concentrated with well-defined edges (not diffuse). The following structures are visible: the malleus (umbo and short process); pars tense (tight portion of the eardrum); annulus; pars flaccida (upper, flaccid fold of the membrane); malleolar folds.

Configuration: flat or concave (not convex).

3. Observe the movement of the membrane, while the subject performs a Valsalva maneuver (forced expiration with the glottis closed), asking him to push; Or if you have a pneumatic otoscope, observe the movement when you inject the air. Movements of the tympanic membrane indicate that the Eustachian tube is patent.

D. Repeat the sequence for the examination of the other ear.

Exploratory techniques of the external and internal structures of the mouth and cranial nerves will be developed when we study the digestive and nervous systems, respectively, in this Section.

NECK EXAMINATION

The neck is explored by inspection, palpation and auscultation. It is always explored sitting. The inspection could be carried out standing, if the subject is shorter than or equal to the examiner.

In your exploration we will take into account:

- Inspection of the neck as a whole.
- Exploration of the thyroid gland.
- Examination of the lymph nodes.
- Examination of the vessels of the neck.
- Exploration of the rest of the structures.

THE NECK AS A WHOLE

When inspecting the neck as a whole, we must explore its shape (short, long, normal), volume (wide, thin, normal), position (central, with lateral deviation, in flexion, in extension) , its mobility, heartbeat, and the presence or absence of tumors.

The exploration of the musculoskeletal structures and the mobility of the neck will be described in this Section, when we address the examination of the osteomyoarticular system, and the heartbeat, in the chapter on the examination of the peripheral vascular system. All alterations

Theological characteristics of the physical examination will be described in Section II.

In addition to the neck regions themselves, the parotid, submandibular and sublingual regions should be explored, as well as the supraclavicular region and the

nape.

EXPLORATION OF THE THYROID GLAND

The thyroid gland is located in the anterior region of the neck (at the junction of the upper two thirds with the lower third): it consists of two lateral lobes joined at their base by an isthmus, which gives it a U" or butterfly shape; and it has an approximate weight of 20-30 g (fig. 4.17). The right lobe is slightly larger than the left. Each lobe is about 5 cm long and 2 cm wide.

The physical examination of the thyroid is performed by inspection and palpation. Normally, the thyroid is not appreciated on inspection and is practically not palpated; If it is of normal size, palpation can only be achieved with special care and with precise rules.

If an increase in volume is detected, auscultation of the gland (presence or absence of murmur in vascularized thyroid) and measurement of the neck should also be performed.

The gland is attached to the trachea and rises when the individual swallows. For inspection and palpation to be most useful, you should also explore it while the subject swallows; Have a glass of water ready for the person to sip when instructed.

It is important to keep in mind that the thyroid gland can be enlarged, although within normal limits, due to different circumstances: country, altitude, puberty, pregnancy, menstruation, etc.; That is, in physiological alarm states of any order, in which it can increase in volume and become more physiologically active.

Inspection

Observe the anterior region of the neck from the front and in profile, if possible with tangential illumination, which can help better detect subtle changes in contour or symmetry. Normally only the glandular isthmus can be seen, especially when swallowing, more frequently in young women.

First, ask the person to keep their head and neck in a normal, relaxed position. Observe if there is any deviation of the trachea, as well as the delimitations of the thyroid and cricoid cartilage, and note if there is any increase in volume (fig. 4.18). Then ask him to extend his neck slightly, tilting his head back, and swallow a sip of water. Observe at that moment the symmetrical upward movement of the

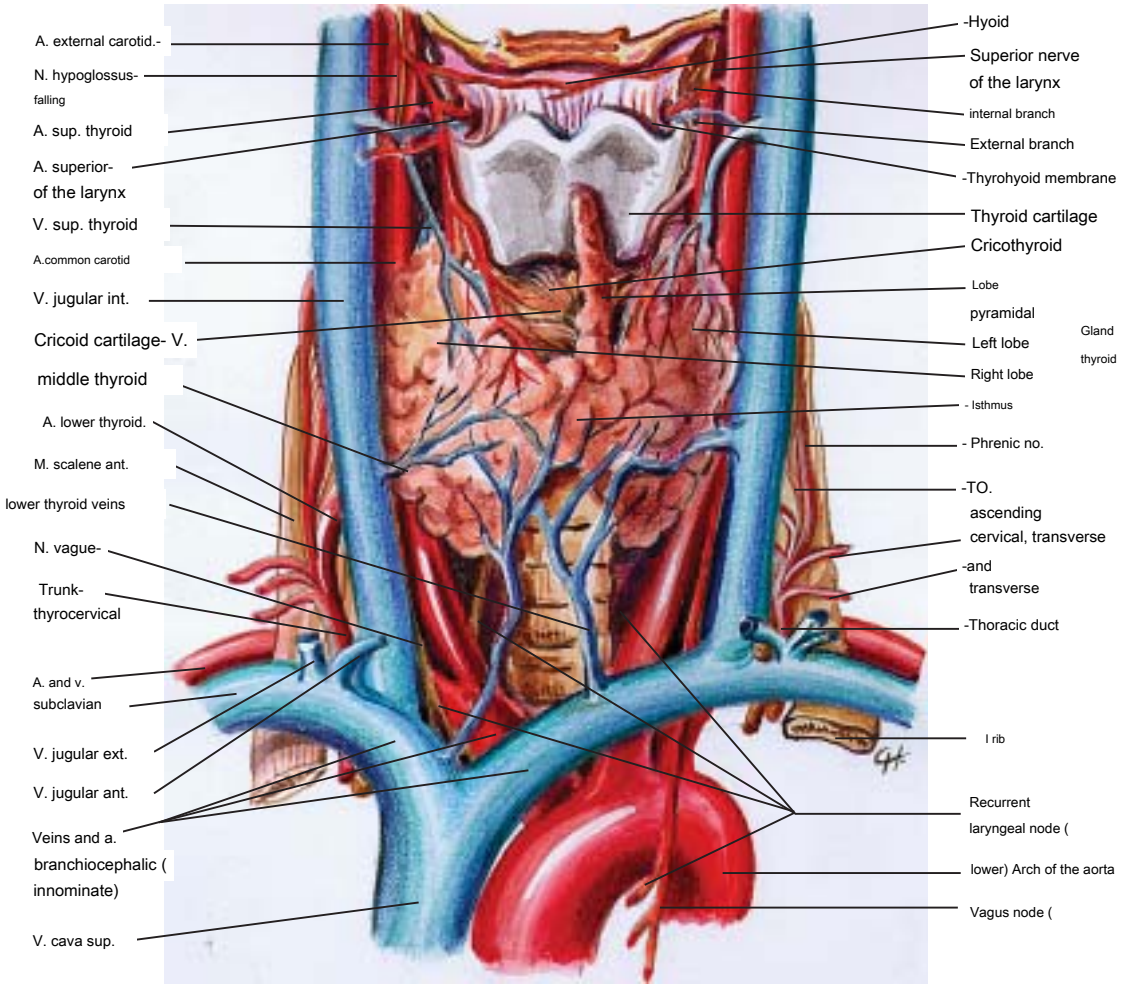


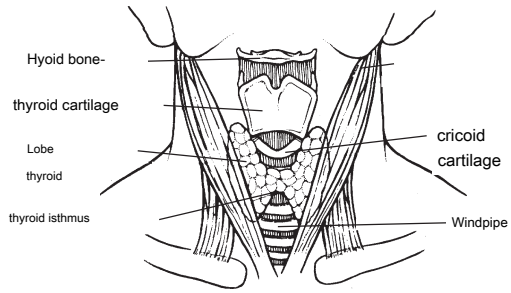
Fig. 4.17 The thyroid gland.

trachea and laryngeal cartilages and, if there is any increase in volume, whether this also moves.

Palpation

When palpating the thyroid gland, one must basically take into account, in addition to the shape and size, already described, its consistency and sensitivity. The gland normally has a rubber-like texture, giving it an elastic consistency upon palpation. Harder masses, in the form of nodules, or those that can be distinguished from their usual texture, are not normal.

Fig. 4.18 Neck structures.



Palpation usually does not cause pain, although the person may experience slight discomfort. Painful thyroid palpation is abnormal, as seen in some forms of thyroiditis.

Palpation is best done with the subject seated, with the explorer standing first behind him, and then in front and on the sides.

Posterior approach

Stand behind the person, who should be sitting with the neck slightly bent, to relax the muscles. Palpation of the thyroid is then performed using the Quervain technique, which consists of surrounding the neck with both hands, with the thumbs resting on the nape of the neck and the four remaining fingers towards the lobes on each side (fig. 4.19).

First place the pads of your index and middle fingers slightly below the cricoid cartilage to locate and palpate the isthmus area. Repeat the maneuver while the person swallows a sip of water, which causes elevation of the isthmus and allows you to further specify its texture, like rubber or elastic. Then ask him

Fig. 4.19 Palpation of the thyroid body using the Quervain technique.

Tilt your head slightly to the left side to palpate the right lobe.

Use the fingers on the opposite side to move the gland laterally, toward the right side, so that the palpating fingers can better feel the lobe. Ask the person to swallow while you examine the lobe. Repeat the procedure on the opposite side.

Previous approach

Stand facing the person, whose neck should be relaxed, but slightly extended, to better expose the underlying gland. Now the hands are placed around the neck, but with the thumbs on the anterior plane, which are the ones that palpate. Palpate the thyroid lobes using the two techniques described below:

1. Facing the subject, the thumb of each hand successively palpates the lobe on the opposite side, looking for nodules (Crile maneuver) (fig. 4.20).
2. Also palpate the lateral lobes, with a variation of the previous technique (Lahey maneuver). It is placed

Fig. 4.20 Palpation of the lobes using the Crile technique, first the right lobe and then the left.

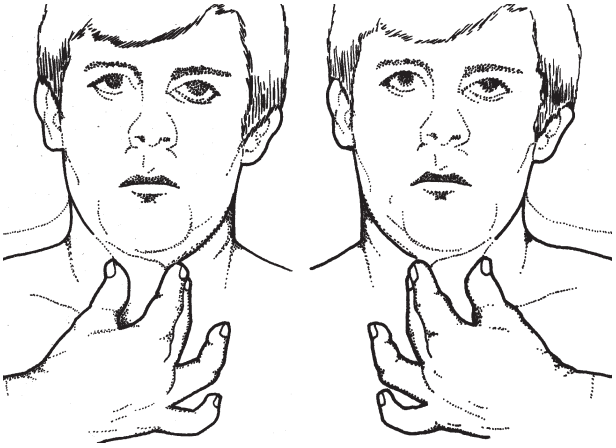


Fig. 4.21 Palpation of the lobes using the Lahey technique.

the pad of one thumb against the lateral surface of the upper trachea, pushing towards the opposite side, so that the lobe of the side towards which it is pushed is externalized more forward and can be more accessible to the thumb of the other hand; This maneuver is completed with swallowing, while palpating (fig. 4.21).

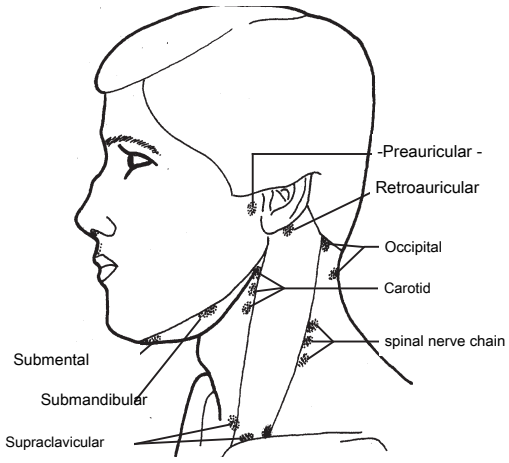
Auscultation

Remember the need to auscultate the thyroid, especially when hyperfunction of the gland is suspected.

EXAMINATION OF THE LYMPH NODES OF THE HEAD AND NECK

The lymph nodes of the head and neck are distributed in lymph node groups; each of them receives tributaries from certain territories. The most important are: preauricular, retroauricular, occipital, submental, submandibular, tonsillar, superficial cervical chain, carotid (internal jugular or deep cervical chain), spinal nerve chain (posterior cervical) and supraclavicular (fig. 4.22).

Fig. 4.22 Groups of lymph nodes in the neck.



General guidelines for the examination and written recording of data

The exploration of the lymph nodes is carried out by inspection and palpation, although the greatest elements are provided by this last method of exploration. Normal lymph nodes are generally neither visible nor palpable.

Inspection

Location of a visible nodule, presence of increased volume or red lines or paths.

Palpation

Palpable lymph nodes are described in terms of location, size (mm or cm), shape, consistency, delimitation, mobility and sensitivity. Determine when the palpable node was first noticed, by the subject or another person.

Technique for the exam

The right and left sides should be examined simultaneously, preferably by a posterior approach and palpating with the index and middle fingers of each hand. Slight bending of the head and turning it away from the area to be examined may be helpful.

Palpate the nodes systematically using a certain sequence, which may be like the following:

1. Pre-earphones (in front of the tragus of the ear).
2. Retroauricular or mastoid (on the mastoid).
3. Occipitals, suboccipitals, or nuchals (at the base of the skull).
4. Tonsillar lymph node (in the angle of the lower jaw).
5. Submandibular (middle distance between the lower mandibular angle and the chin).
6. Submental (in the midline, behind the tip of the chin).
7. Superficial cervical chain (over the sterno-cleidomastoid muscle).
8. Posterior cervical chain (anterior to the trapezius muscle).
9. Deep cervical chain (embedded in the sternocleidomastoid muscle). It is difficult to palpate. To do this, hook your thumb and index finger around the sternocleidomastoid and then palpate.
10. Supraclavicular (within the angle formed by the sternocleidomastoid and clavicle).

Palpable lymph nodes

Small palpable lymph nodes are common. Palpable nodules less than 1 cm wide, circumscribed, mobile, and non-painful, are often considered benign, but such findings should be recorded. You may detect an infarcted lymph node, associated with chronic or frequent inflammation. An infarcted lymph node is enlarged by more than 1 cm, is mobile, painless, hard and nodular. Malignant diseases can produce palpable nodes that are characteristically non-painful, non-movable (fixed to the underlying tissues), irregular in shape, and of a firm, rubbery or elastic, or nodular consistency. Such findings require further examination in greater depth.

Written record of findings

Example 1. As lymph nodes are generally neither visible nor palpable, the examination result should be recorded as follows: "lymph nodes not visible or palpable in the body area or specific lymphatic chain. No pain on palpation, no edema, no skin color changes."

Example 2. Benign palpable lymph node: "Non-palpable neck nodes except the right tonsillar node, 1.5

The venous vessels of the neck are explored primarily through inspection and the arterial vessels by inspection, palpation and auscultation.

As most of the information in the examination of the neck is obtained through inspection and fundamentally, palpation, the beginner tends to forget the auscultation of the neck, both the thyroid and the trachea, as well as the vessels of the neck. For details on the examination, see examination of the peripheral vascular system in this Section.

EXAMINATION OF THE REST OF THE NECK STRUCTURES

In addition to the organs already described, the larynx and trachea are explored in this region. When examining them, their position and movements must be noted.

The techniques for exploring osteomyoarticular structures and neck mobility will be described in the SOMA examination chapter, in this Section.

CHEST EXPLORATION

In this chapter we will only detail the physical examination of the arms and armpits, and the breast self-examination. The remainder of the chest examination will be discussed in the chapters.

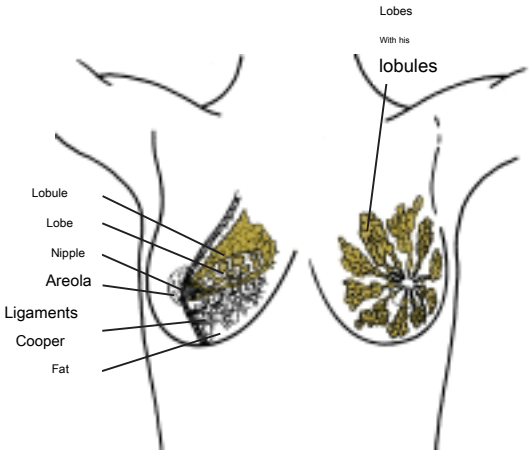


Fig. 4.23 Structure of the female breasts.

titles corresponding to the physical examination of SOMA, respiratory and cardiovascular, in this same Section.

PHYSICAL EXAMINATION OF THE BREAST AND ARMPITS

The examination of the breasts and armpits is the same for men and women. The discussion is oriented toward female breasts, because women have more breast tissue.

Brief anatomophysiological account

The breasts are modified sebaceous glands, located one on each side, between the second and sixth ribs and between the sternal border and the mid-axillary line, on the anterior wall of the chest. In the center of each breast is the nipple, surrounded by the areola.

The breast is made up of glandular tissue, fibrous tissue and fat, which causes the most varied breast sizes. The amount of glandular tissue is similar in all women and is organized into around twelve to fifteen lobes. These lobes are made up of lobules that contain the acini, the milk-producing glands. Each lobe has a duct that ends in the nipple (fig. 4.23).

The breasts contain fibrous tissue or Cooper's ligaments, which attach them to the skin and the fascia of the pectoralis muscle.

Although the breasts are usually symmetrical, a slight variation in shape between them is normal. Breast size can vary with age, menstrual cycle and pregnancy.

Glandular breast tissue is found in large concentrations in the upper outer quadrant of each breast. From this quadrant, a small portion of breast tissue, known as Spence' s tail, extends towards the axilla (fig. 4.24).

Areolas are pigmented areas that surround each nipple. Its color can vary from pink to brown and can change during pregnancy. Sebaceous glands or Montgomery tubercles are found in the areola. The nipples are projections of tissue that contain the ducts from the milk-producing glandular tissue.

The breasts contain some groups of lymph nodes (Fig. 4.25). Most of the lymph drains into the armpit. The internal lymphatic system close to the breasts frequently serves as a vehicle for the spread of cancer.

General guidelines

The breast examination has two components: inspection and palpation, carried out with the person in a sitting and lying position. For inspection, the chest and breasts must be completely exposed. During the breast examination, you must provide appropriate explanations, in a way that instills confidence, does not violate the principles of medical ethics and can avoid iatrogenesis and embarrassing situations, both for you and the person being examined. If your hands are cold, try to warm them beforehand by rubbing them against each other. Cold hands can interfere with comfort

Fig. 4.24 Spence's tail.

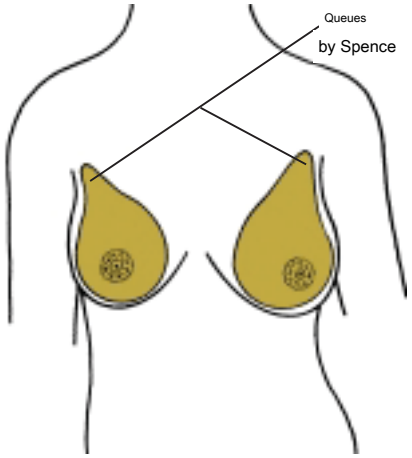
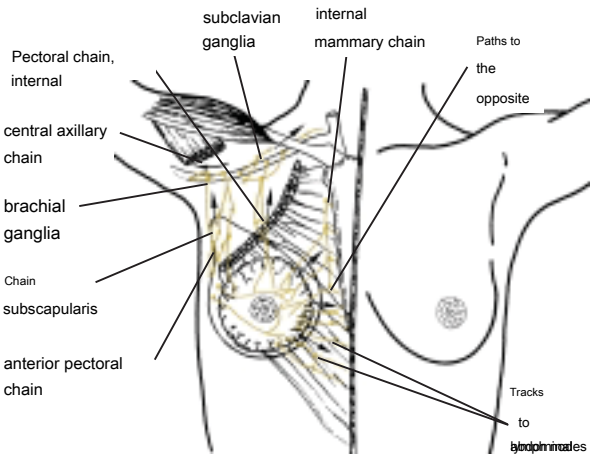


Fig. 4.25 Lymphatic drainage of the manma.



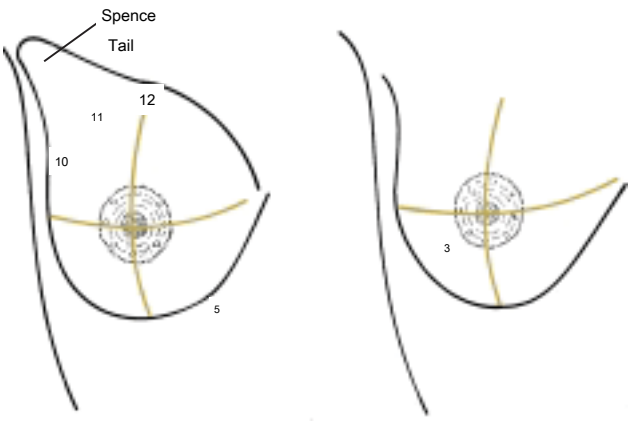


Fig. 4.26 Limits of breast examination.

of the patient and with the interpretation of the findings of the exam.

Delimitations of the mother for the exam

The breast can be described using two mapping methods: the hourly or clock method and the quadrant method.

In the hourly method, the breast is compared to the face of a clock, with the nipple in its center. Lesions or other findings should be located by their position on a clock face, such as at 6' or 2'. The quadrant method divides the breast into four areas:

upper internal, upper external, lower external and lower internal. The horizontal and vertical lines intersect at the nipple (fig. 4.26).

male breasts

The examination of male breasts is essentially the same as that of female breasts. The breast should feel flat and smooth on palpation and no glandular breast tissue should be present. The size and shape of male breasts varies according to body structure and fat distribution. Gynecomastia is the increase in glandular breast tissue in the male breast. Gynecomastia has multiple causes, such as drugs, liver or endocrine disease, but it is also observed during pubertal changes. This condition should receive prompt attention, especially if the increase is a recent development. The armpit is evaluated the same in men and women.

Guidelines for examination and written recording of data

- Size, shape, symmetry.

-Color, texture, lesions and vascular patterns of the skin.

Fabric quality.

- Lymphatics of the breast.

- Discharge from the nipple.

Exploratory techniques

Moms

Perform the breast inspection with the person in five different positions:

1. Sitting with arms at the sides of the body.
2. Sitting with arms raised above the head.
3. Sitting, leaning forward.
4. Sitting with your hands pressing your hips.
5. Lying down.

Inspection in these five positions allows you to evaluate the quality of fibrous tissue within narrow limits, to determine if any part of the breast is fixed, and to detect asymmetry or decreased mobility with changes in position.

A. Inspect the breast with the person in a sitting position.

Arms at the sides of the body:

Ask the person to sit comfortably with their arms at their sides. Observe the breasts for symmetry, size, shape, skin color, texture, vascular patterns, presence of moles and visible nodules. Next inspect the areola to determine its size, shape, and surface characteristics. Also observe the direction and symmetry of the nipples and if there is any discharge.

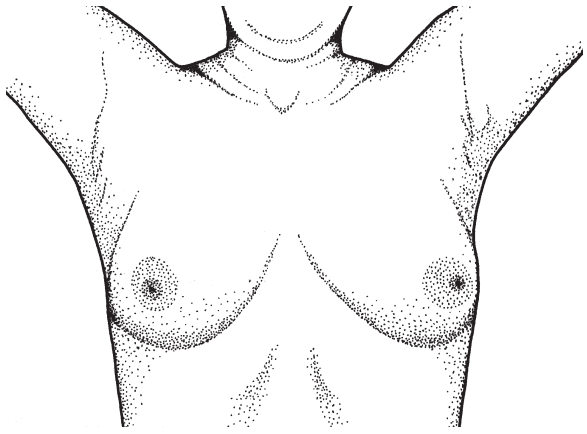
If you detect any abnormalities, ask the person when the finding was first noticed, and if there have been previous evaluations or treatments. Inspect the breasts from different angles.

Arms raised above the head (fig. 4.27):

Ask the person to raise their arms above their head and continue inspection of the breasts.

The size and shape of the breasts should remain symmetrical during movement. Write down the exact position

Fig. 4.27 Examination with arms raised.



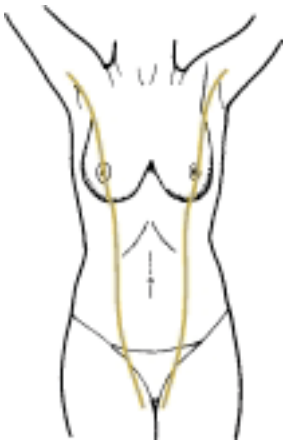


Fig. 4.28 Breast lines.

ta of some supernumerary nipple (multiple congenital nipples, found along the mammary line, from the armpit to the groin) (fig. 4.28).

Leaning forward (fig. 4.29):

Ask the person to lean forward. To do this, you may need to hold her arms to help her balance. If you prefer, she can stand and lean forward. Continue inspection.

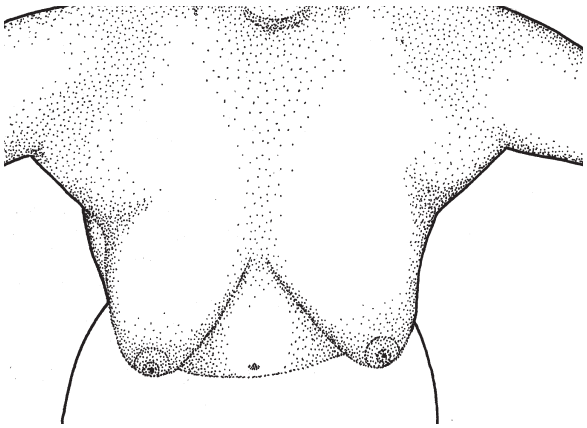
Pressing the hands on the hips (fig. 4.30):

Ask the examinee to place their hands on their hips and press to contract the pectoral muscles.
An alternative method is for the person to put their hands together and press against each other. Inspect as before.

B. Palpation with the person sitting.

Palpation of the breasts should be done in the supine position. The sitting position is optional, unless the person reports an abnormal finding, has a history of breast abnormalities, is at high risk of breast cancer due to family history, or has pendulous breasts.

Fig. 4.29 Leaning forward.



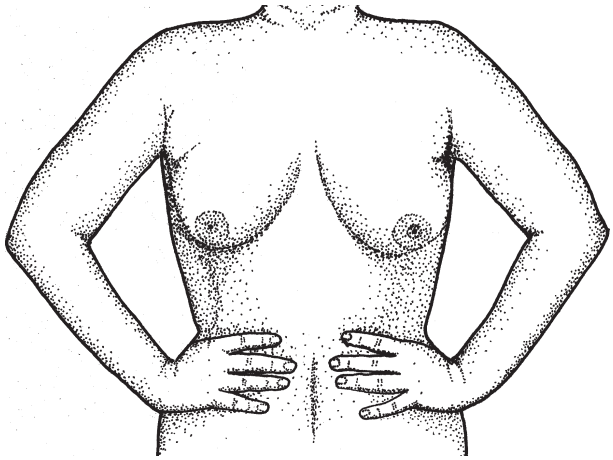


Fig. 4.30 With hands on hips.

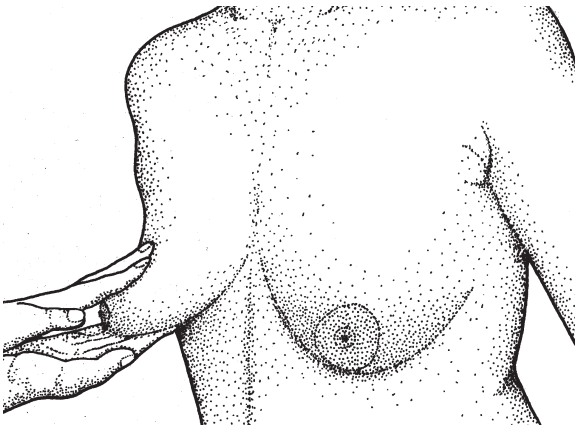
Palpate the pendulous breasts bimanually. Use one hand to support the underside of the breast while palpating with your dominant hand, moving from the chest wall toward and including the nipples (Fig. 4.31). Notice if the nipple appears to be fixed to the wall, or shortens or enlarges with movement.

Palpation of the armpits

The palpatory areas include the following lymphatic chains: lateral subscapular, central axillary, subclavicular and supraclavicular (from outside to inside and from bottom to top). Most of the lymphatic drainage from the breast is toward the armpit. Palpation of axillary lymph nodes is abnormal.

Palpate the armpit when the muscles are relaxed. To relax your muscles, adduct your forearm and place it on top of yours. Place the hand of the forearm that is supporting the person's forearm in the armpit and the dominant hand on the anterior surface of the chest.
Locate the lymph nodes in the armpit, according to their anatomical position, and evaluate them by gently rolling the tissue under your fingers against the wall.

Fig. 4.31 Bimanual palpation.



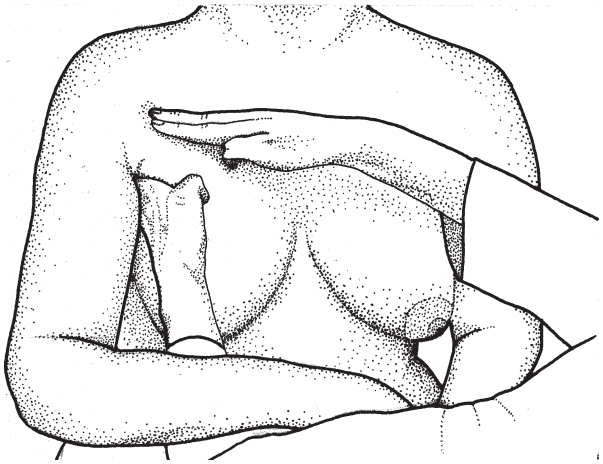


Fig. 4.32 Sitting axillary palpation.

thoracic. Examine the anterior, posterior, medial and lateral aspects of the axilla (Fig. 4.32).

Palpate the anterior surface bimanually, trying to access the nodes near the pectoralis muscle.

Also palpate the subclavicular and supraclavicular nodes.

culars.

C. Inspect and palpate the breasts with the person lying down (see procedure for inspection with the woman sitting).

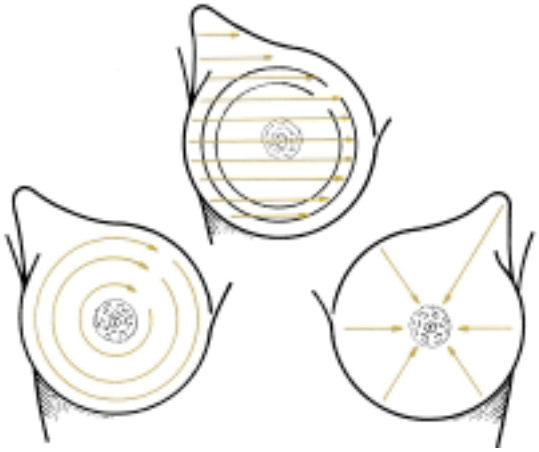
1. Lie the person on the examination table.

Place your forearm on the same side of the breast that you are going to palpate, above and behind your head, comfortably. For women with moderately large or large breasts, place a folded towel or small pillow under the shoulder on the upper back to further shift the tissues.

Proper positioning shifts the breast tissue further onto the chest wall.

2. Perform superficial palpation, followed by deep palpation, to evaluate breast texture and detect masses.

Fig. 4.33 Methods to palpate the breast.



Palpate all four quadrants, including the superolateral extension of the breast, called Spence's tail.

When palpating, move your fingers in a circular motion, without abandoning it until the palpation is complete.

exam.

Use a systematic method that ensures a complete and consistent evaluation. Begin firm palpation, in the same place on both breasts. Use the clockwise method, the horizontal line method (inside out horizontally from top to bottom), or the radial method (periphery to center) (fig. 4.33).

Pay special attention to the upper outer quadrant and Spence's tail, as most breast cancers develop in this area.

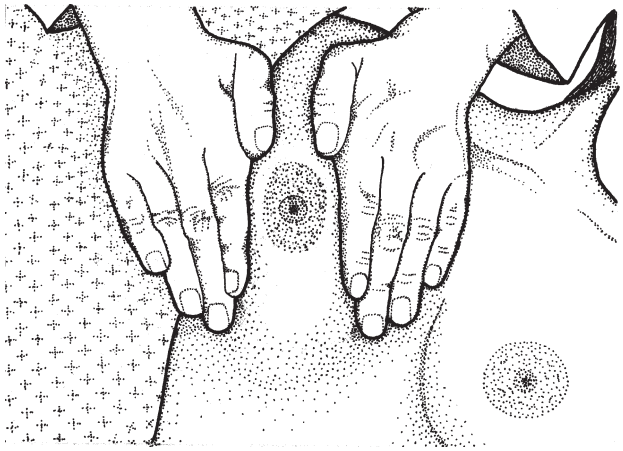
3. Palpate the areola for underlying masses and compress the skin around the nipple to evaluate masses and secretions. If you notice any secretion, compress the breast along the suspicious ducts to identify the lobe that produces the secretion (fig. 4.34) .

Interpretation of the findings

A. Normal inspection findings.

The shape and size of the breasts can vary according to body type, but they should be symmetrical. A very slight asymmetry may be normal, if it is not of recent appearance. The nipples should look the same on each breast and point slightly upward and laterally. Inverted nipples may be congenital and the finding is insignificant if it existed before puberty. However, this can cause difficulties with lactation.

The skin color should be the same on each breast and similar to the skin on the abdomen. The areolas should be Fig. 4.34 Looking for discharge from the nipple.



pink or brown. The nipples and Montgomery tubercles may be slightly darker than the areola.

Normal vascular patterns in the skin are asymmetrical. During pregnancy, an increase in vascularization can be seen, indicated by a bluish or reddish hue. Such vascular changes are symmetrical and diffuse. The skin must be free of lesions; However, moles and nevi are common. The breasts are susceptible to the same injuries as the rest of the skin.

Deviation from normal to inspection:

A unilateral change in the shape, size or symmetry of the breast is abnormal. When the arms are raised or lowered, when the patient lies down or leans forward, any change in symmetry may be the result of a mass or injury that restricts the ability of the ligaments to stretch. Observe whether the nipple appears to be fixed to the wall or shortens or enlarges with movement.

B. Normal findings on palpation.

The breast tissue should be smooth, elastic, soft and easily movable. During palpation the normal breast feels glandular and lumpy. The lumpy sensation is a result of the glandular tissue of the lobes, fat, and connective fibers.

Generally the breast is not uniform in quality, but the two breasts are symmetrically uniform. In the premenstrual period the breast may feel more lumpy and sensitive. An accumulation, which is normal, may be noticed at the positions between 4 and & on the clock. The nipples should be smooth and may become erect during palpation.

There should be no nipple discharge in a non-hormonally stimulated breast. The secretion can be normal during pregnancy, lactation, sexual stimulation of the breast and as a side effect of some psychotropic drugs.

Written record of findings

If any breast enlargement is discovered, record the following characteristics:

- Location, following clockwise or quadrants and the distance in centimeters that separates it from the nipple.

- Size of the find, in centimeters.

- Shape and contours.

-Consistency (soft, hard, rubbery or elastic, stoney).

- Mobility.

-Color of the skin that covers it.

- Sensitivity.

- Some sign of retraction.

If any lymph nodes are palpated in the axillary area, record the following:

- Location.

- Size.

- Shape or contour.

-Consistency.

- Mobility.

- Sensitivity.

Carefully record the examination findings so that comparison with subsequent findings is realistic and reliable. Any changes reported by the person must also be recorded.

BREAST SELF-EXAMINATION

Every woman over 20 years of age should perform a breast self-examination monthly, according to the recommendations of the American Cancer Society, since nearly 90% of breast nodules are detected by women themselves or their peers.

The findings of breast self-examination must be validated by a health professional, who must also perform a periodic examination, at least every three years before age 40 and annually after age 40.

Men should also have breast exams as frequently as women.

In addition to a physical breast exam, women should have a baseline mammogram between ages 35 and 40, a mammogram at least every two years between ages 40-49, and an annual mammogram after age 50.

Guide to performing breast self-examination

It is important that the student, from their first steps in the clinic, learn how to perform breast self-examination, so that they can teach it and practice it, in the case of belonging to the female sex.

Examine the breast at three different times:

1. In front of a mirror, to detect unusual contours or changes in the appearance of the skin, such as wrinkles, depressions or depressions and retractions of the nipple.

Observe the appearance of the breasts, in three positions:

a) Arms at the sides of the body (fig. 4.35).

b) With arms raised, above the head (fig. 4.36).

c) Hands on the hips, while contracting the chest muscles (fig. 4.37).

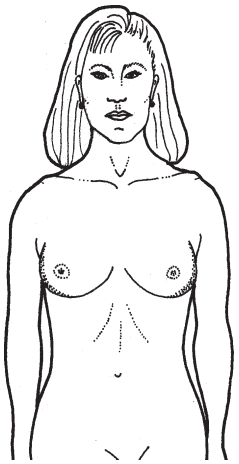


Fig. 4.35 Arms at the sides of the body.

2. During bathing, standing in the shower or sitting in the bathtub, when the skin is damp and the hands move easily over the breast (figs. 4.38 and 4.39):

a) Use your right hand to examine the left breast, while raising your left arm above your head to expose more breast tissue.

b) Do the opposite for the examination of the right breast.

3. Lying down:

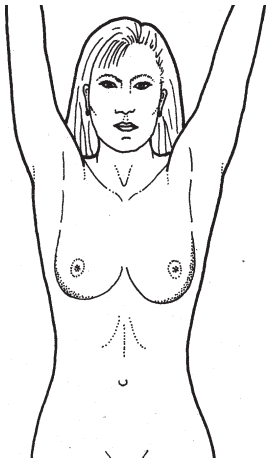
a) Place a small pillow, cushion or blanket under the shoulder on the side to be examined, with the arm up, the elbow flexed 90° and the forearm or hand above or below the head, for better exposure of the breast tissue (fig. 4.40).

b) Use your left hand to examine the right breast and vice versa.

c) Be meticulous, palpating with your fingers, in a circular manner, from the center to the periphery. Also palpate the breast tissue that extends to the armpit.

d) Finally, squeeze the nipple to detect if there is any discharge (fig. 4. 41).

Fig. 4.36 Braz0S high.



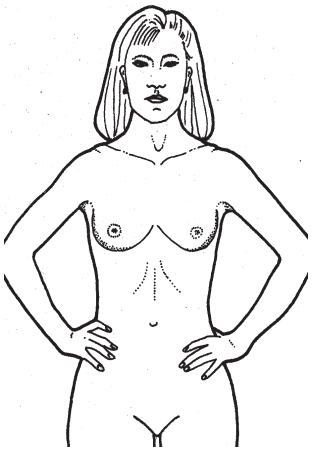


Fig. 4.37 Hands on hips.

If there is any skin change, hard lump, or clear or bloody discharge from the nipple, you should tell your doctor immediately.

High risk groups

Breast cancer is the second leading cause of death in North American women. The Occurrence increases with age and is almost eight times more common in women between 75 and 80 years old than at ages between 35-40 years.

Women at risk for breast cancer include the following:

- Over 40 years of age.
- Those who have never been pregnant or those whose first full-term pregnancy occurred after age 33.
- Those with early menarche (before age 12) or late menopause (after age 50).
-Women with a history of benign breast disease.
- Those with a family history of breast cancer (mother, sisters) .

Fig. 4.38 Self-examination in the shower.

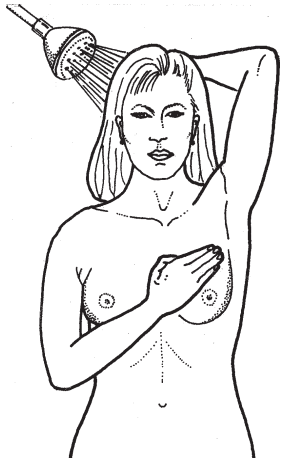
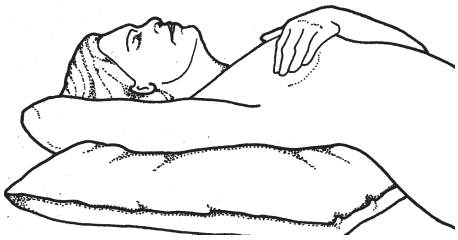


Fig. 4.39 Self-examination in the bathtub.

Fig. 4.40 Breast self-examination lying down.



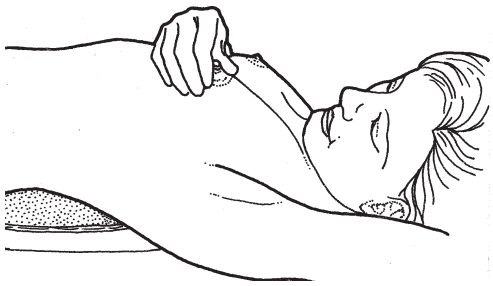


Fig. 4.41 Squeezing the nipple.

SPINE AND LIMBS

The techniques for exploring the spine, extremities and joints will be developed in the chapters corresponding to the exploration of the osteomyoarticular and peripheral vascular systems.

ABDOMEN EXPLORATION

Exploration of the abdomen requires special attention and will therefore be treated in the next chapter of this Section.

REGIONAL PHYSICAL EXAM

OF ABDOMEN

In this chapter we will study the abdomen as a whole, as a unit, applying the classic exploration methods: inspection, palpation, percussion and auscultation. The combined physical methods will be studied in Section II, in the chapter on the digestive system, corresponding to the alterations of the physical examination of the abdomen. Below we briefly present the necessary knowledge of applied anatomy.

SUMMARY OF DESCRIPTIVE AND TOPOGRAPHICAL ANA TOMY

The internal limits of the abdominal cavity are: above, the diaphragmatic dome; below, the upper inlet of the pelvis; behind, the lumbar spine, and in front and on both sides, the abdominal wall itself, which is made up of muscles, aponeurosis, subcutaneous cellular tissue and skin. The abdominal wall has its internal surface covered by the peritoneum (parietal), a serous layer that reacts easily to any visceral injury.

The external limits of the abdomen are: above, the costal margin, from the base of the xiphoid appendage to the seventh dorsal vertebra (D7), the so-called thoracoabdominal line; Below, it is limited externally by another line that extends from the crural arches, passing through the iliac crests, to the fourth lumbar vertebra (L4), called the abdominopelvic line. This lower limit is rather artificial, since the abdominopelvic cavity must be considered simultaneously, which is an ovoid with a diaphragmatic major pole and pelvic minor pole.

CLINICAL ANATOMY

It is essential to apply anatomical knowledge to the clinic, taking into account how practical it is to project the different abdominal viscera and their areas and points of greatest symptomatic relief on the wall.

AREAS OF THE ABDOMEN

In order to carry out the external projection of the abdominal viscera and their areas, the French or Latin school has devised the division of the

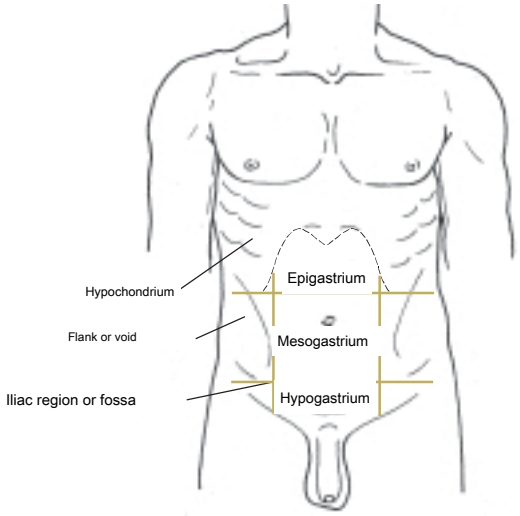


Fig. 5.1 Zones of the anterior wall of the abdomen according to the French school.

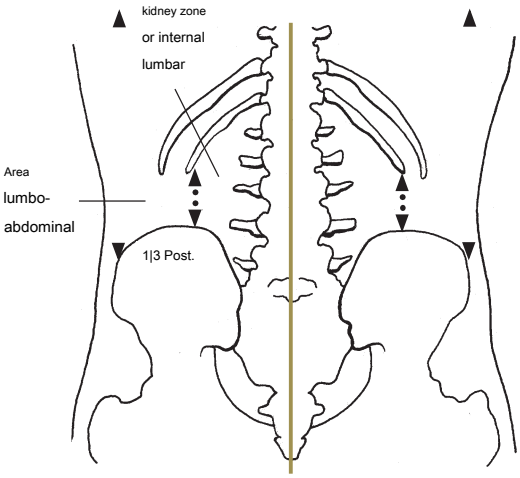


Fig. 5.2 Zones of the posterior wall of the abdomen according to the French school.

outer wall of the belly in nine zones at the expense of a layout constituted as follows:

In front, two ascending vertical lines are drawn that start from the external end of the horizontal rami of the pubis and reach the anterior ends of the X ribs. Two other horizontal lines are crossed, the upper one, which joins the right In this way, a somewhat irregular board is obtained, which contains in its area the costal ridges, the xiphoid appendage and the lower lateral portions of the costal grills. This board presents the nine most important abdominal visceral projection zones (fig. 5.1).

In the upper portion and in the center, we have the epigastrium;
on the sides, the right and left hypochondria, the latter correspond in their external portion, almost entirely, to the hard parts (last ribs); but deeply, the upper lateral portions of the abdominal cavity.

In the middle portion, in the center, we have the mesogastrium or umbilical region and on the sides, the voids or right and left flanks.
In the lower portion we have, in the center, the hypogastrium and on the sides, the right and left iliac fossae.

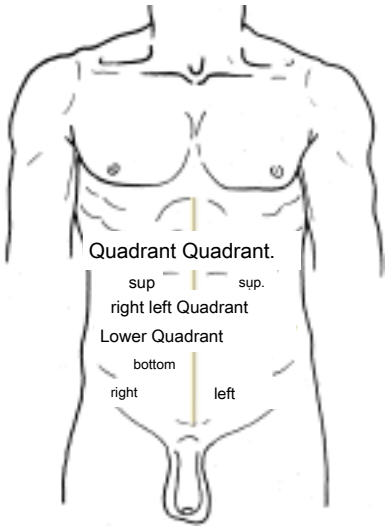
Behind it there is another layout, at the expense of two lateral vertical lines that go from the XII rib to the posterior third of the iliac crest, which with the vertebral midline form four more zones (fig. 5.2).

Inside, on the sides of the spine and limited by the conventional lines described, we have the internal lumbar or renal zones: outside these lines conventional vertical areas, the external lumbar or lumboabdominal areas, which continue without precise limits forward with the flank areas, described on the anterior wall.

The Anglo-Saxon school simplifies the clinical anatomy of the abdomen by using only four large areas, formed by true quadrants (fig. 5.3), at the expense of drawing two conventional lines: one mid-vertical and the other horizontal, which intersect exactly at the navel.

It is necessary for the doctor to know both systems of clinical anatomy, since the descriptions and locations of symptoms in medical literature can equally refer to one system or the other.

Fig. 5.3 Division of the abdomen according to the Anglo-Saxon school.



VISCERAL PROJECTIONS BY ZONE

We will refer to the content of the nine topographic zones described by the French school (fig. 5.4), since the knowledge of the visceral projections in the abdominal quadrants of the Anglo-Saxon authors is obvious.

1. Epigastrium. Left lobe of the liver; a portion of the anterior surface of the stomach with part of the body, the antrum and the pylorus; gastrohepatic omentum with the hepatic artery, the portal vein and the cystic and common bile ducts; Winslow hiatuses; second and third portions of the duodenum; pancreas; superior mesenteric artery; solar plexus and vertebral column with the aorta, vena cava and thoracic duct.
2. Right hypochondrium. Right lobe of the liver:; bottom of the gallbladder; part of the transverse colon and hepaic flexure; upper extremity of the right kidney and adrenal capsule.
3. Left hypochondrium. Left lobe of the liver; greater gastric tuberosity; cardia; gastrosplenic omentum; spleen; upper extremity of the left kidney and adrenal capsule; small portion of the descending colon and splenic flexure; loops of the jejunum and tail of the pancreas.
4. Mesogastrium or umbilical area. Greater omentum; lower gastric portion; transverse colon; loops of the small intestine; mesentery; cava and aorta.
5. Void or left flank. Part of the small intestine and left colon.
6. Empty right. Part of the small intestine and right colon.
7. Hypogastrium. Greater omentum; part of the small intestine; bladder and ureter, as well as the uterus in women.
8. Left iliac fossa. Sigmoid; lower portion of the descending colon; thin handles; genitals in women; iliac and psoas vessels.
9. Right iliac fossa. Cecum and appendix; thin handles; psoas; genitals in women; ureter; iliac vessels.

In the posterior region, we have: lumbar regions (internal or renal) that include the right and left kidneys, the renal pelvis and the beginning of the ureters.

GENERAL PHYSICAL EXAMINATION OF THE ABDOMEN AS A

In the study of the general physical examination of the abdomen as a unit, we will successively develop the semitechnics of the abdomen, that is, the study of the different methods and maneuvers used in the inspection, palpation, percussion and auscultation of the abdomen, considering

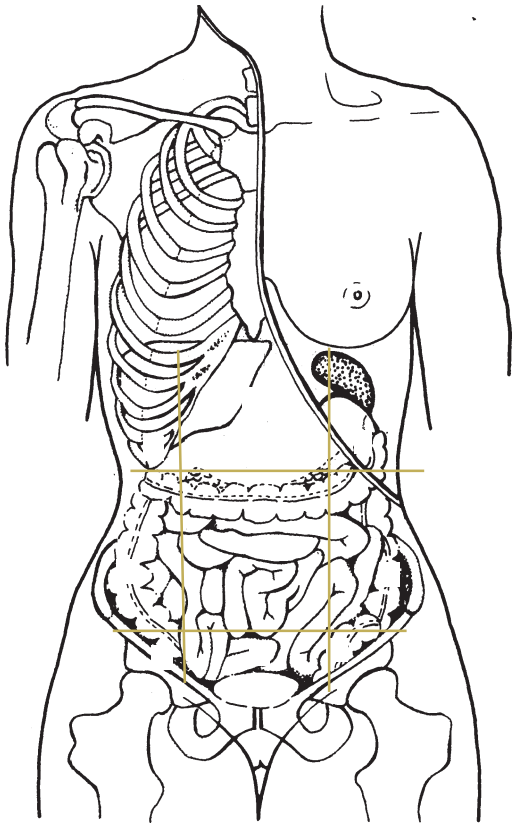


Fig. 5.4 Visceral projection by zones of the anterior wall according to the French school: 1, epigastrium; 2, mesogastrium; 3, hypogastrium; 4 and 5, hypochondria (right and left); 6 and 7, empty (right and left); 8 and 9, iliac fossae (right and left).

derated as a whole the normal data obtained through examination in presumably healthy subjects (making the pertinent observations according to the constitutional types).

EXPLORATION TECHNIQUES

Although basic examination techniques are generally used in the sequential order of inspection, palpation, percussion, and auscultation, the abdomen should be examined in the following sequence: inspection, auscultation, percussion, superficial palpation, and deep palpation. It is always auscultated first, because percussion and palpation can alter intestinal air-fluid sounds. The diaphragm of the stethoscope is used to auscultate intestinal sounds, which are usually high-pitched, and the bell is used to auscultate vascular sounds, which are low-pitched. Before deep palpation perform superficial palpation.

Percussion and palpation can be combined. When examining one organ you should do so completely, by both methods, before proceeding to examine another organ. For example, when examining the liver, first percuss its boundaries and then palpate it.

Before beginning the examination, the subject's bladder must be empty. Ask the person to urinate; This way we make sure that a full bladder is not confused with a pregnancy, a tumor or a cyst.

Because many examinees may be expectant and anxious about the exam, the procedure must be explained to them to gain their confidence, avoid questions, and relieve their anxiety.

The person should be lying face up (supine or dorsal position). The explorer will use a bed, or a table, almost rigid, on which the subject will have his body fully supported, the thorax and head placed at a level slightly higher than the abdomen or at the same level, the arms extended to the sides of the body or crossed over the thorax, and the legs parallel, with the lower limbs extended in very slight flexion, to obtain the greatest physical rest, and therefore, the maximum relaxation of the abdominal muscles. Many people tend to put their arms up, generally supporting their head, and/or cross their feet, which should not be allowed, as this contracts the abdomen and makes the examination more difficult.

Many teachers teach their students that the bed should be completely horizontal and that the pillow should be removed to avoid abdominal contracture. In practice we observe that when the subject lies completely straight and without a pillow, neck extension and abdominal contracture are caused.

Place a small pillow on your head so that it is at the same level as your chest and prevents neck extension, but does not cause it to bend. If possible, place another one under your knees for more complete relaxation.

If it is a sick person, the examiner must also take into account the patient's symptoms, since if he or she has shortness of breath at the time of the exam, it is very likely that this will increase when lying completely horizontal.

Sometimes in specific maneuvers, the subject will also have to adopt other positions: lateral decubitus, ventral position, special positions, as well as the standing position.

The examiner should stand on the right side, if it is right, because many of the special examination techniques relate to the liver and other structures on the right side, and using the right hand positioned on this side makes the maneuver easier.

Before starting the exam, ask the person if there is any abdominal area in which they feel discomfort or pain. These areas must be examined last and the patient must be assured that the existing discomfort will not worsen, or that it is essential to cause some discomfort for a short time, in order to arrive at a diagnosis, so their cooperation is necessary.

Even if the subject is not in pain, encourage him or her to immediately express pain or discomfort during the exam. Follow the patient's facial expression and body language. Be especially receptive to facial expressions of pain, discomfort, or anxiety to try to determine what action precipitated the change in expression.

Keep the patient warm during the exam to avoid abdominal tension. Also, the exam room, your hands, and the stethoscope should be kept warm. If necessary, rub your hands against each other to warm them, and the stethoscope against one of your hands, with a cloth such as a sheet, or by placing or rubbing it on your forearm for a few seconds.

Examine the entire abdomen systematically following the same order, to get used to the normal sensations in each area. It is logical to think that if the patient has already reported the presence of pain of a defined location, one should not start in that area, but rather in the place as far away as possible from the painful area. If there is no precise pain, a systematic examination method could be: starting from the left iliac fossa, advance in a spiral, counterclockwise, to the left flank, left hypochondrium, epigastrium, right hypochondrium, right flank, right iliac fossa, hypogastrium, to end in the umbilical region and thus, the entire abdomen will have been covered in the first instance.

menu

This is fundamentally applicable to percussion and palpation, but can also be used for auscultation. Another variant of the systematic examination method used for palpation is to begin palpating the left iliac fossa with the right hand, also going up through the void or left flank to the hypochondrium on that side and palpating the epigastrium. Now we go down to palpate the mesogastrium, then the other hypochondrium, then we go down through the right void to the iliac fossa on that side and, finally, we palpate the hypogastrium. Use whatever variant is most comfortable for you, but get used to always using the same one, to get the most benefit.

ASPECTS TO BE REQUIRED IN THE EXAM AND IN THE WRITTEN RECORD

Inspection: contour, symmetry, respiratory movements, pulsations, peristalsis, skin integrity,

masses.

Auscultation: intestinal sounds (fluid sounds), vascular sounds.

Percussion: tone, limits of the abdominal organs.

Palpation: muscle tone, characteristics of the organs, sensitivity, masses, pulsations, fluid accumulation.

Inspection

The belly should be widely uncovered and if possible very naked, to observe the abdomen in its relations with the neighboring regions, but the chest and inguinal regions should be covered, while these are not being examined, to avoid discomfort and respect modesty.

Sometimes, the abdominal inspection should also be performed with the subject standing and perhaps, on particular occasions, in a sitting or other special position.

The abdomen must be observed from the front and in profile, to do so, all angles or incidences of light rays on the abdominal surface are taken advantage of. The examiner must perform the observation, standing, if the subject lies on a high table: sitting, if he or she lies on a low bed.

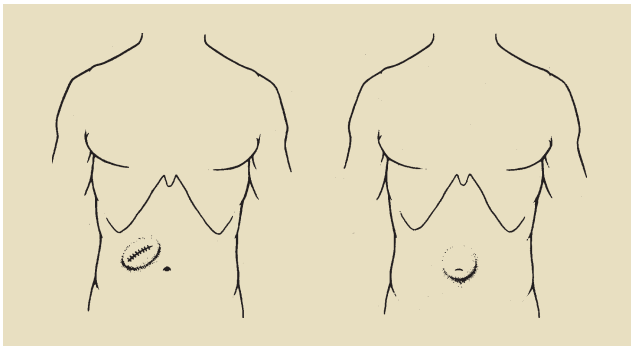
Observe, whether standing or sitting on the right side of the patient, the volume, shape, relief, symmetry, masses, pulsations, the integrity of the skin and the respiratory pattern, whose data must be inspected, both in the static order with complete relaxation, and in the dynamic order, inviting the patient to breathe and move in different positions, sometimes looking for abdominal contracture. Ask the patient to cough to reveal pain with cough and protruding masses (hernias), not previously seen (Fig. 5.5).

Sitting or leaning, look at the abdomen tangentially to evaluate its contour and peristaltic movements.

The normal abdomen is symmetrical, on either side of its midline. There are no visible volume increases. In thin individuals, pulsation of the abdominal aorta in the midline and peristaltic movements can be seen. There should be no skin lesions, except surgical scars. Stretch marks may be found if the skin has been stretched, as in pregnancy, obesity, abdominal tumors, and Cushing's disease. Respiration is abdominal in men (the abdomen rises and falls with breathing).

The altered contours of the abdomen are:

Fig. 5.5 Profusion produced by hernias: a, incisional hernia; b, umbilical hernia.



1. Generalized distention with an inverted navel: obesity, gas distension.
2. Distention of the lower half: distended bladder, pregnancy, ovarian mass.
3. Generalized distension with everted navel: ascites, tumor, umbilical hernia.
4. Excavated abdomen: emaciation, replacement of subcutaneous fat with muscle.

Auscultation

It is performed to identify normal and pathological abdominal sounds. The same lying position in which the subject is found is used. Immediate (almost discarded) and mediate auscultation can be performed, either with the simple stethoscope, obstetric type, or with the binaural stethoscope, in common use.

Listen for bowel (airborne) sounds with the diaphragm of the stethoscope. It is not necessary to hear in each quadrant if the pitch and frequency are normal. If the sounds are hypoactive or absent, listen to each quadrant for one to two minutes. Normal hydroaerial noises are high-pitched, burping sounds, with a regular frequency between 5-35/min.

Use the bell to hear vascular sounds and abdominal rubs. Vascular sounds and rubbing are not normal findings.

Percussion

Percussion of the abdomen, in general, is much less important than palpation. It is performed with the patient lying face up and, by exception, in other positions. Percussion attempts to identify the different abdominal sounds, which normally depend on the more or less solid nature of the intra-abdominal viscera. The technique followed is that of Gerhardt or digit-digital. Percussive blows spread easily in the abdomen, therefore, it is recommended that they be performed extremely gently.

We will not describe in detail this method, which has already been well studied. In this chapter on general abdominal percussion, specific organ percussion techniques are excluded, which we will learn about later, in the chapter corresponding to the examination of the digestive system.

The percussion is done covering the entire belly, with the purpose of having an overall impression. Systematically percuss the abdomen in all quadrants to evaluate for abnormal sounds. The predominant sound is tympanism; dullness is obtained on the masses and solid abdominal organs. Tympanic sound indicates gas in the underlying intestine.

Palpation

In the abdomen, palpation is of utmost importance, because there are many and very useful objective symptoms that can be collected. At the same time, it is not easy at all, since everything depends on the manual skill of the explorer and his preparation (talent and reasoning) to interpret the perceived sensations.

Mimicking Boas, we will say that it is necessary to "palpate while thinking and think by palpating."

In the first moments of training the skill, you will have to think about the correct execution of the technique, step by step; But you should not lose sight of your learning that the ultimate goal is the assessment of what is being examined. So try to simultaneously incorporate the thought of what you are looking for and the result of that search.

On the other hand, one should not be abrupt or violent in palpation, because when hurt, a reflex contraction and fear are triggered, which will prevent a good examination.

Below we will make some general considerations about abdominal palpation, before its systematic analysis.

Let's say that by placing one hand, or both, on the belly and manipulating the fingers in a variable way, the abdominal wall is depressed and mobilized and in this way, a series of sensations are collected that inform more or less about the normal or pathological state of the wall, as well as the organs included in the abdominal cavity.

Without exaggerating the imaginative note, so as not to fall into the "virtuosity" that was cultivated in another era by Haussmann, ObratzOW and others (in which there were no other complementary resources), many physical symptoms of great use in the diagnosis can be obtained roughly.

Palpation with conscious perception of what is touched is achieved by the stimulation of receptors that correspond respectively to superficial and deep sensitivity. Palpating, especially with the pads of the fingers, excites the Meissner corpuscles, which are involved in superficial sensitivity.

The deeper-lying Paccini corpuscles, Golgi corpuscles and muscle spindles are the elements excited for deep sensitivity, which is obtained by palpating with the palm of the hand and fingers, and at greater pressure than that used to acquire superficial sensitivity.

The contact of the hands and fingers with the abdominal wall and its contents can be made with more or less pressure, approaching the wall with the hand flat, or in an oblique way to try to penetrate the cavity and obtain sensations of resistance or consistency; or with the maneuver of unevenness, tracking or sliding.

In general, when palpating, one finds: the abdominal wall itself with its constituent elements, as well as the superficial organs (upper face and anterior border of the liver, small intestine, colon and pylorus) and the deep organs (pancreas, aorta, ovaries, kidneys and angles of the colon). The gallbladder may appear superficial or deep.

Following Lombardi and Vitale, from the Argentine school, let us consider not precisely superficial and deep palpation, but the terms of palpation of the abdominal wall or continent (generally superficial type) and palpation of the intracavitary organs or the contents (generally deep type), although, sometimes, an organ can be palpated at the expense of a superficial palpation, and the wall, at the expense of a deep palpation.

Finally, there is the particular sensation of the so-called abdominal tension or degree of resistance of the abdomen as a whole, in which elements of superficial and deep sensitivity intervene, which is why it is analyzed in particular.

Having made these general considerations about palpation, let us successively analyze the requirements that must be met for its performance:

- Requirements inherent to the examinee.
- Requirements regarding the browser.
- Maneuvers applicable in cases of palpation that is apparently impossible due to parietal hypertonia.
- Palpation of the abdominal wall or continent (superficial palpation).
- Abdominal tension.
- Visceral, intracavitary or content palpation (deep palpation).

Requirements inherent to the examinee

The subject should be lying down with as much abdominal relaxation as possible, as explained at the beginning. Some doctors prefer that the person flex their legs and even their thighs over the abdomen, but sometimes this helps the abdomen contract more than it does to relax. Any movement of the subject, when nervous (of the head, limbs, etc.), causes muscle contractures. Breathing should be calm and broad, with the chest elevated to help relax the abdomen.

Despite these instructions, it may happen that when the examiner begins palpation, sometimes only with the gesture of moving his hands, the patient, by reflex, starting from an affected site, a painful area or strictly as a psychological starting point, establishes a state of partial or global hypertonia, which makes palpation difficult or impossible.

If the subject is very sensitive and suffers from "tickling" or is very anxious about the procedure, begin by palpating with the subject's own hand, under yours. Later we will see other maneuvers applicable in these circumstances.

Browser Requirements

We insist that the doctor, or any explorer, should preferably be placed to the right of the patient, sitting at an appropriate height from the bed or stretcher, or standing if the patient is standing. Your arms and forearms should form an obtuse angle, not a straight or acute one, since in these latter positions the movements of the hands would be forced and could cause muscle contractures that would make perception difficult. You must act delicately to gain the subject's trust and prevent him from getting nervous; your hands should have a comfortable temperature; If necessary, you can heat them previously; nails must be trimmed. You should "palpate", not " puncture" or apply annoying or counterproductive pressure.

Maneuvers applicable in cases of apparently impossible palpation due to parietal hypertonia

Cover the skin of the abdomen with Vaseline or talcum powder to facilitate manual sliding.

Use the Galambos method, which consists of depressing the abdomen with one hand, at a distance no greater than 10 cm from the area you want to explore with the other hand (fig. 5.6).

- Apply local anesthesia (Lemaire), or general anesthesia.
- Perform hypnosis on the patient.

Palpation of the abdominal wall or continent (superficial palpation)

Systematically palpate each quadrant or area of the entire abdominal wall with single-manual palpation to detect pain, muscle spasm, or tumors.

Fig. 5.6 Galambos method.

Single-manual superficial palpation consists of the use of one hand, the right or the left depending on the examiner; The palm of the hand is gently placed on the abdomen, with the fingers extended and joined, to exert gentle pressure on the wall, with downward movements with the tips of the fingers, trying to depress the abdomen by about 1 cm. This is how you explore the different portions of the belly and get your first overall impression. It can be palpated with the hand, in plane, helping with the fingers in an oblique or penetration plane, as well as with the ulnar or radial edge (fig. 5.7).

Encourage the patient to breathe slowly and deeply, and exhale with the mouth open, while palpating. The abdomen should feel relaxed with the exhalation. Muscle contraction can indicate anxiety. Also notice the facial expression of the examinee.

Through this operation, it is captured whether or not there are alterations in the different planes that make up the wall. Possible localized vaulting or masses may be explored that, if they belong to the wall, persist when the patient sits, breathes in, or raises his or her head or feet.

To do this, while palpating the mass, ask the patient to raise his head and shoulders, as if trying to sit up. If the mass remains palpable, it is in the abdominal wall. The muscle tension caused by this maneuver does not allow the intra-abdominal masses to be palpated. An alternative method may be to perform palpation of the wall, with the lower limbs raised, resting on the skin of the bed, or on any surface that keeps them elevated (fig. 5.8).

The thickness of the wall is explored by pinching it with the fingers (fig. 5.9); The skin, cellular tissue and even the muscles are examined, if the patient has a very thin wall.

The rectoabdominal muscle, which includes two large central muscles, should be palpated on the sides of the

Fig. 5.7 Single-manual palpation.

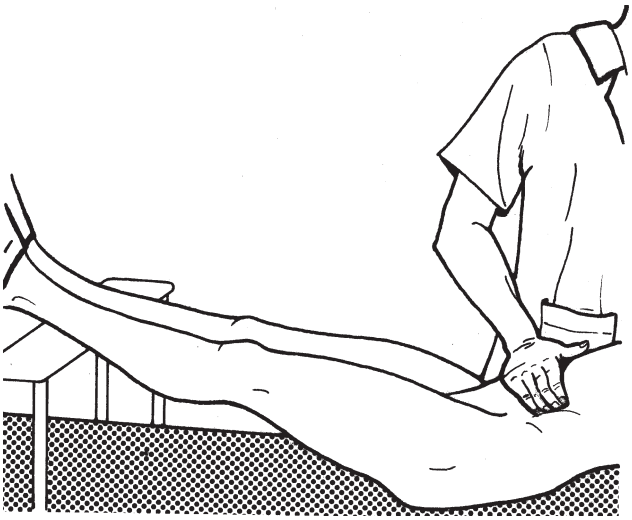


Fig. 5.8 Exploration of the abdominal wall.

midline and united in it, which extend from the xiphoid process to the pubic symphysis.

On superficial palpation, these abdominal muscles should feel relaxed, rather than contracted or spastic.

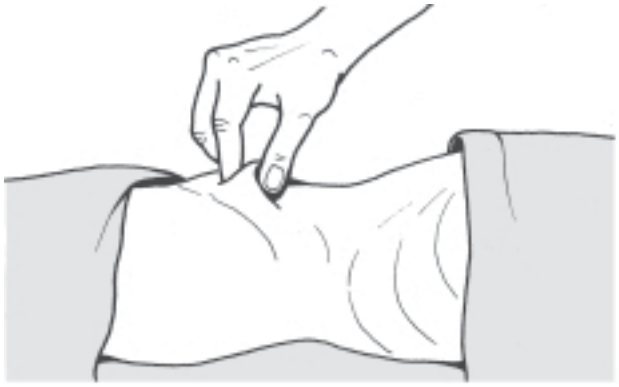
The natural orifices, possible solutions of continuity of the wall and the abdominal midline are also explored. Palpating the midline, a separation of the rectoabdominal muscle can sometimes be noticed when the person raises the head, especially in obese or pregnant people. It may be palpable as a midline groove, but this does not represent a significant problem.

Also in the midline or at the level of surgical scars, possible incisions are explored with the fingers and edges of the hands.

In addition, the umbilicus and the orifices in the inguinal regions should be especially palpated, an area that is often forgotten to inspect and palpate and which can lead to costly diagnostic errors.

If it is found that something is protruding, it is detailed by palpation and reintegrated, if possible (reduction of hernias), into the interior of the abdominal cavity.

Fig. 5.9 Exploration of skin thickness.



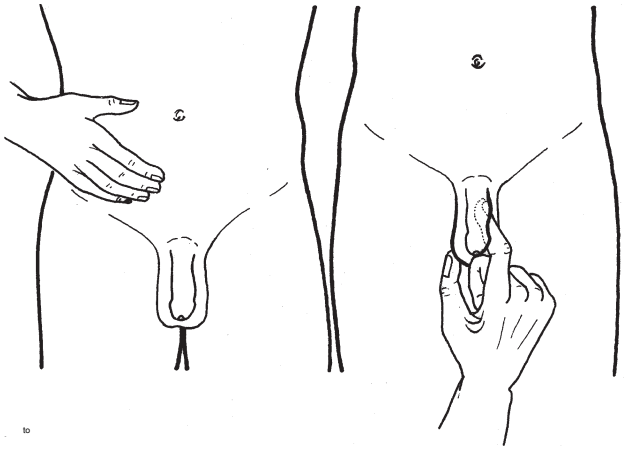


Fig. 5.1O Inguinal palpation looking for hernias: a, abdominal palpation; b, palpation of the annulus.

The orifice is palpated, detailing its characteristics (fig. 5.10 a), and the patient is made to cough or push, to cause possible organ leakage through the hernia orifices.

Special palpation of the inguinal ring looking for hernias (Fig. 5.10 b) is performed later, during the examination of the genitals, and is therefore described later, in the corresponding chapter of this Section.

Finally, at this stage of palpation, the sensitivity of the integuments and the superficial pain caused should be explored.

The verification of alterations in parietal sensitivity will be done by gently pulling or pinching the skin, while palpating its thickness (see Fig. 5.9), or passing over it a pin (Fig. 5.11), edge of the nail, cotton ball, etc. The patient's reaction can be verbal or through facial expression.

Carnet, who has particularly studied the important role of the abdominal wall in the examination, recommends, in order to verify whether pain caused in the wall depends on some intracavitary organ, that the patient raise the head or feet or project the wind.

Fig. 5.11 Examination of parietal sensitivity: the skin is explored with a pin.

go out. In these circumstances, pain and the hypertonic reaction should increase, if they are determined by an alteration of the abdominal wall. In all these cases of parietal pain, this author carefully explores the spinal column and possible postural defects of the subject.

abdominal tension

To explore this phenomenon of balance between the intracavitary pressure determined by the distention of the viscera and the resistance of the wall that tries to contain it, the right hand is palpated in different places of the belly, placing it flat and using the fingers to exert some pressure against the wall. The hand must always be oriented parallel to the axis of the body and this palpation can be done in different positions of the subject being examined: lying on his back, on his side, standing, etc. Normal data varies in different areas and will be described with the normal data obtained in the physical examination of the abdomen.

Visceral, intracavitary or content palpation (deep palpation)

During this stage of abdominal palpation, which is the fundamental one, the subject remains lying in a supine position; The lateral decubitus position is used if you want to bring an organ to the midline, thus making it more superficial and more mobile. The standing or sitting position favors the descent of the viscera; The patient should be invited to lean the trunk forward to seek greater relaxation.

Visceral palpation varies depending on breathing, so the doctor must know the physiological influence of breathing (movement of the diaphragm and viscera) in this sense.

On inspiration, the diaphragm lowers, intra-abdominal pressure increases and palpation becomes difficult. During expiration, the opposite happens, palpation is easier and more useful. The viscera slide under the fingers during respiratory movements, if the respiratory type is costoabdominal, which should always be sought, since it is the most appropriate. Sometimes it is advisable to make the patient breathe deeply to cause the liver to descend during inhalation, for example, and project this organ further into the abdomen. As can be seen, the examiner can vary breathing as desired, for better visceral palpation.

Visceral or intracavitary palpation can be performed with one hand (monomanual) or with both hands (bimanual). Single-manual palpation has already been explained previously.

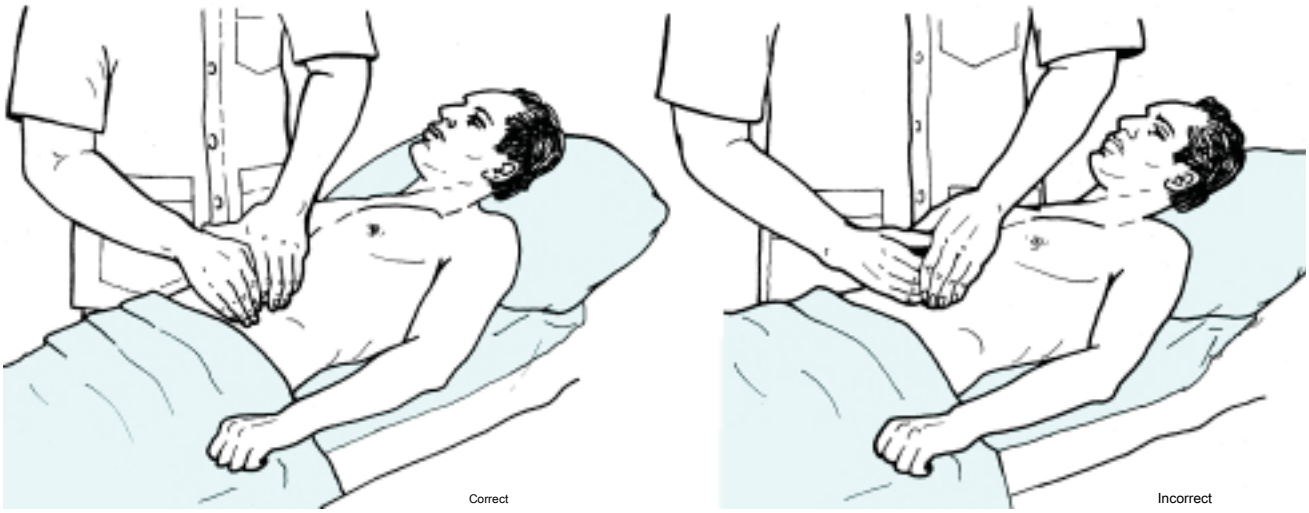
Bimanual palpation is much richer in variety and provides greater wealth of data. We will describe the general types of bimanual palpation most used and will leave the special palpation maneuvers for each organ for the particular physical examination.

Types of bimanual palpation

1. With index fingers in contact.
2. With overlapping hands.
3. Anteroposterior or rally.
4. With the sliding, uneven or dragging maneuver.

In bimanual palpation with the index fingers in contact, it is preferable that the hands diverge at their bases, to take advantage of the finger pads more freely (fig. 5.12). The hands must have a certain degree of obliquity (a double obliquity) on the abdomen to gain the necessary depth for palpation of the viscera when practicing the glide.

Fig. 5.12 Bimanual palpation with the index fingers in contact.



Correct Incorrect

Fig. 5.13 Bimanual palpation with overlapping hands.

Bimanual palpation with overlapping hands (fig. 5.13) aims to overcome a certain resistance of the wall, although in reality it is palpated with only one hand, the bottom one.

Bimanual anteroposterior or rally palpation, used by Chauffard (Fig. 5.14), is more applicable to voids. With this palpation, the hand behind pushes the viscera forward, offering them to the palpating hand. In all these types of palpation, the fingers play a fundamental role; They should be handled with ease, not fixed in a forced, flexion or true claw position, which is detrimental to good palpation. In turn, two, three or even four fingers of each hand should be used.

Finally, following Haussmann, let us remember the need to practice the maneuver of sliding the fingers over the organs, which is facilitated by dragging the skin over the tips of the fingers to leave the excess skin folded there (fig. 5.15). In this way it deepens from top to bottom; movements are taken advantage of

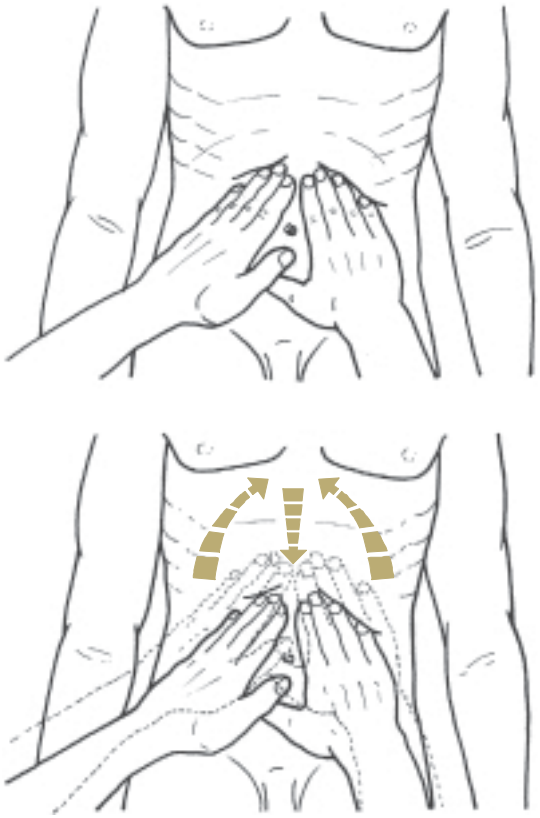


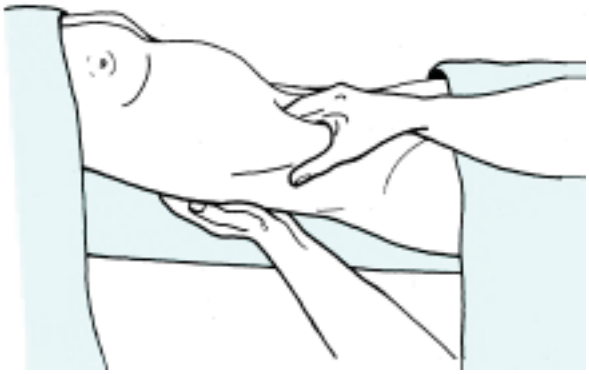
Fig. 5.15 Finger sliding maneuver.

respiratory movements and thus the fingers, when sliding, palpate the desired segments and appreciate the possible alterations.

It is always advisable to do the sliding to show the difference in levels, placing the hands perpendicular to the largest diameter of the anatomical formation being palpated.

This maneuver is mainly used for palpation of the liver and spleen, which are located more superficially and below and in contact with the diaphragm.

Fig. 5.14 Bimanual anteroposterior or rally palpation.



Operations to perform deep palpation

1. Monomanual or bimanual palpation can be used with the index fingers in contact.

   Using single-handed palpation and with the same hand position as for superficial palpation, palpate deeply the abdominal quadrants, but avoid pricking your fingers with your movements; use the entire flexor surface of the fingers, depress them all at the same time, with a flexor movement of the metacarpophalangeal joints.

2. Again encourage the examinee to breathe slowly and deeply, and exhale with the mouth open, while palpating.

3. If the abdomen is difficult to depress, repeat the palpation, placing one hand on top of the other (bimanual palpation with hands overlapping).

The fundamental objectives of deep palpation, or better, intracavitary, are the assessment of palpable masses and the existence or not of pain, not previously detected with superficial palpation.

Palpable masses. Although it is not normal to find palpable masses, if they are found, their location, size, shape, consistency and mobility must be evaluated. The location should also include whether the mass is located in the abdominal wall or is inside the abdominal cavity.

Abdominal structures that are commonly misinterpreted as abnormal masses include the aorta, the rectoabdominal muscle, the feces-filled colon, the sacral promontory in thin people with too deep palpation, and the pubic symphysis in obese people with redundant abdomen. The feces are palpated usually as a tubular structure, as opposed to a rounded structure of an abnormal mass.

Pain. Although the abdomen is not normally painful, some people report pain on deep palpation, especially over the abdominal aorta, cecum, and sigmoid colon.

If the person manifests abdominal pain from the beginning, rebound pain is also explored by depressing the abdomen and quickly withdrawing the fingers upward, a technique that will be detailed in the Propedeutics Section. Rebound pain may indicate inflammation of the peritoneum.

Palpation of intracavitary organs also includes special palpation of the liver, spleen and kidneys, the techniques of which will be discussed in each of the corresponding chapters of this Section.

MODEL OF WRITTEN RECORD OF THE NORMAL ABDOMINAL EXAMINATION

Example of a normal abdominal exam, as part of your annual checkup. The exam was recorded as follows:

Inspection: flat, symmetrical abdomen, with well-developed muscles. No visible masses or pulsations, no pain or protrusions with spontaneous breathing or provoked cough; intact skin, without lesions.

Auscultation: active, audible and normal hydroaerial sounds in all quadrants. No vascular sounds.

Percussion: loudness of the abdomen as a whole, normal.

Palpation: not painful on superficial or deep palpation, no palpable masses.

OSTEOMIOARTICULAR SYSTEM (SOMA).

CLINICAL ANATOMY AND PHYSIOLOGY

6

Adequate exploration of the osteomyoarticular system (SOMA) is not exclusive to rheumatologists and orthopedics, but is a necessity for every doctor, since this system intervenes in practically all the voluntary functions of the individual, as exponents of the relationship life and therefore is constantly subject to external and internal factors, capable of producing its affectation.

Before beginning the study of SOMA exploration, it is essential to make an anatomophysiological description of it.

CLINICAL ANATOMY AND PHYSIOLOGY

The SOMA is composed of bones, muscles, tendons, joints and other periarticular structures.

We must make each of the components of this system independent for a better understanding in the didactic order.

BONES

The bones make up the skeleton; They are the part of the osteomyoarticular system where the muscles are inserted through the tendons. Its function in movement is to serve as a lever when muscle contraction is carried out. They are made up of osteoblasts and osteocytes and a calcareous intercellular substance.

Osteoblasts are the original cells of the bone system and osteocytes are housed in osteocytic cavities or lacunae; Its cells are fusiform and have branches that anastomose with other neighboring cells (fig. 6.1).

Collagen fibers are the main component of the intercellular substance; They are surrounded by a substance rich in mucopolysaccharides, mainly chondrointin sulfuric acid. Among its constituents we also find mineral salts, especially tricalcium phosphate.

Bone tissue is constantly formed and reabsorbed by a substance generated by osteoblasts, which calcifies immediately (fig. 6.2).

The process of mineralization of bone substance requires the presence of calcium and phosphorus in high concentrations and an enzyme, alkaline phosphatase. Osteoclasts are responsible for resorption.

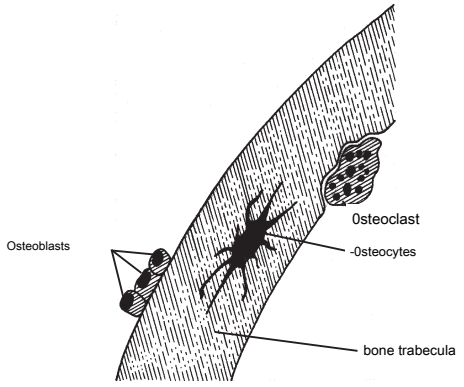
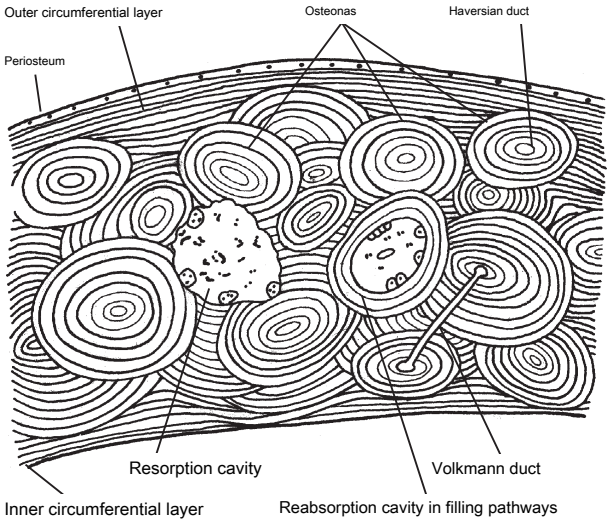


Fig. 6.1 Bone tissue cells.

Fig. 6.2 Renewal of bone tissue.



Bone tissue can be spongy or compact, its distribution differing depending on whether the bone is flat, long or short. Structurally, the bone has three parts:
the diaphysis, with a central cavity where the spinal cord is located; the metaphysis, between the diaphysis and the epiphysis, separated by the connecting cartilage and made up of spongy bone tissue and covered by the cortex; At the end is the epiphysis, very close to the articular cartilage (fig. 6. 3).

The bone marrow forms blood cells during childhood; In the adult this function is reduced to the pelvis, proximal parts of the femur and humerus, vertebrae, ribs, sternum and bones of the skull.

MUSCLES

A muscle can be defined as an organ composed of bundles of contractile fibers that perform movement. There are three types of muscles: smooth, skeletal and striated.

From the SOMA point of view, we are interested in the study of skeletal muscle, because it is the one with the widest distribution in the economy and because it is

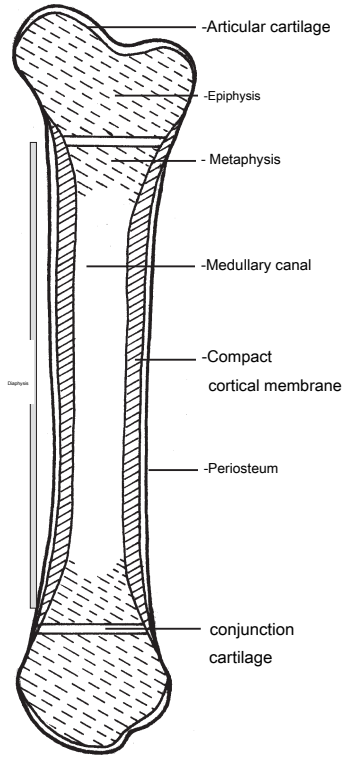


Fig. 6.3 Constitution of a long bone.

attached to the bones, making movement possible, which can be reflex or voluntary. By striking the leg a little below the kneecap, she jumps; The same thing happens when you kick a ball, but in the first case it is a reflex movement and in the second it is voluntary.

The muscle is composed of large polynuclear cells, attached to the sarcolemma or external membrane. The cytoplasm or sarcoplasm contains numerous mitochondria.

Striated muscle is so called because it is made up of dark and light bands, located at the same level of each muscle fiber. The muscle fiber presents the motor plate, where the nerve endings from the motor nerve that innervates them arrive, forming the subneural apparatus of Contaux, which releases an enzyme, called cholinesterase. In the muscle there are also sensory receptors that react to both pain and muscle stretching (fig. 6.4).

The muscle fibers are grouped and form fascicles, wrapped and partitioned by a conjunctiva membrane. This membrane has different names: epimysium, if it surrounds the muscle; perimysium, if it surrounds the fascicles, and endomysium, if it is between the muscle fibers (fig. 6.5).

The blood content of the muscle decreases during contraction. The muscle contraction is maximum from its beginning, responding to the "all or nothing" law (Starling's Law) , that is, a muscle fiber can be in a condition of relaxation or total contraction; There are no intermediate stages.

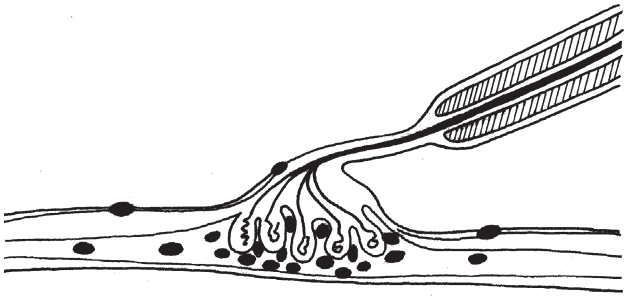


Fig. 6.4 Myoneural junction.

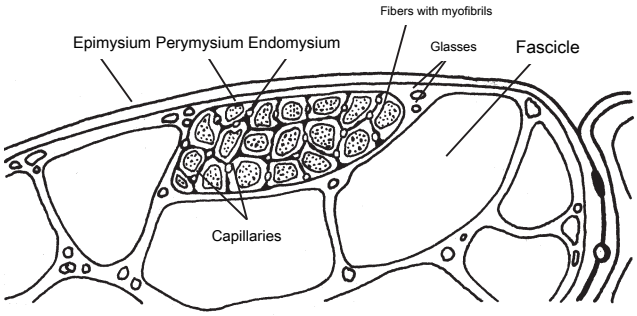
The anterior horn of the spinal cord sends out its motor extensions, dividing into a certain number of branches that innervate each muscle fiber individually at the level of the motor plate. The set of muscle fibers innervated by a nerve cell forms a myoneural branch, so the nervous excitation of the same motor unit forces the simultaneous contraction of all the muscle fibers dependent on it, that is, it constitutes a synchronous activity (fig. 6.6).

Muscular activity is governed by electrical and biochemical phenomena. The muscle contraction is triggered from the motor plate by a depolarization wave that runs through the fiber and causes a very brief action potential that is replaced immediately afterwards, leaving the fiber in a position to reproduce the same phenomenon. This impulse sent by the motor branches produces, at the level of the motor plate, the release of acetylcholine, which initiates the depolarization of the muscle fiber, causing contraction. Acetylcholine is immediately destroyed by cholinesterase, repolarizing the fiber that is ready to restart the process.

The striated muscle contracts or shortens by virtue of various stimuli: thermal, mechanical, chemical, but usually muscle activity is a response to nervous activity.

Muscle is made up of adenosine triphosphate (ATP), phosphocreatine and glycogen. ATP releases energy, giving rise to muscle activity and converting first into adenosine diphosphate (ADP), and subsequently into adenosine monophosphate (AMP) (Fig. 6.7).

Fig. 6.5 Cross section of a muscle.



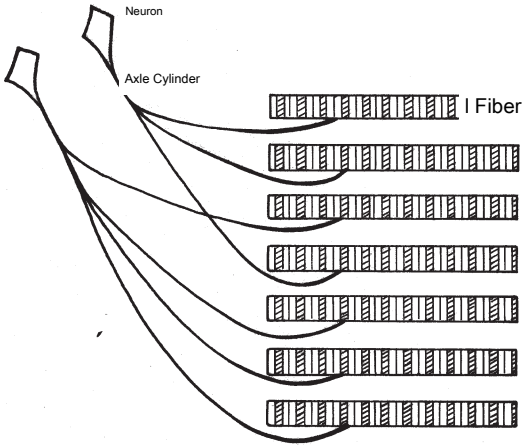


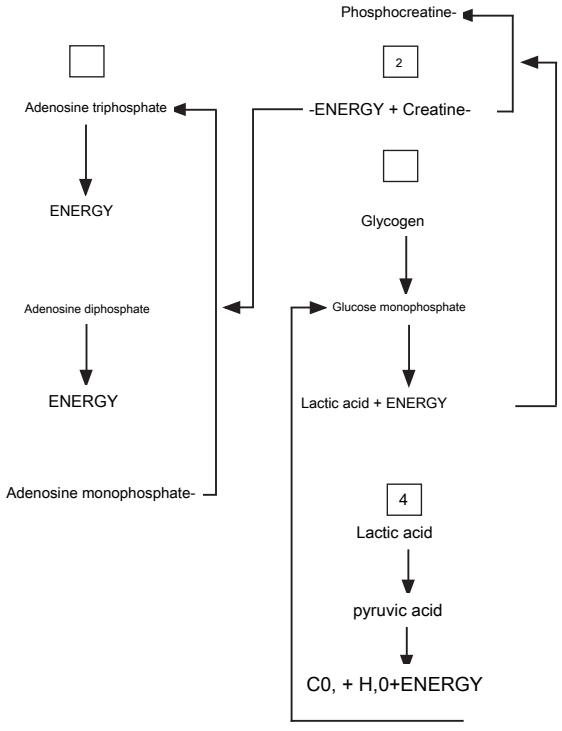
Fig. 6.6 Drive unit (pm: drive plate).

Phosphocreatine is broken down into energy by phosphate and releases creatine; This energy is used to resynthesize ATP. Glycogen is transformed into lactic acid through a phosphorylation process, also releasing energy, which is used in the resynthesis of phosphocreatine. In this process of muscle contraction there is great consumption of oxygen, with the release of carbon dioxide.

TENDONS

The muscle is inserted into the bone through tendons. The union between the tendon and the bone is very powerful, to the point that an abnormal effort can damage the muscle, but rarely the tendon.

Fig. 6.7 Chemical phenomenon of muscle contraction.



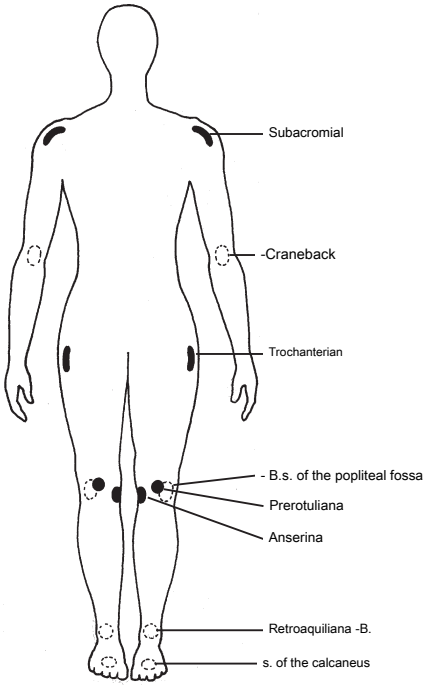
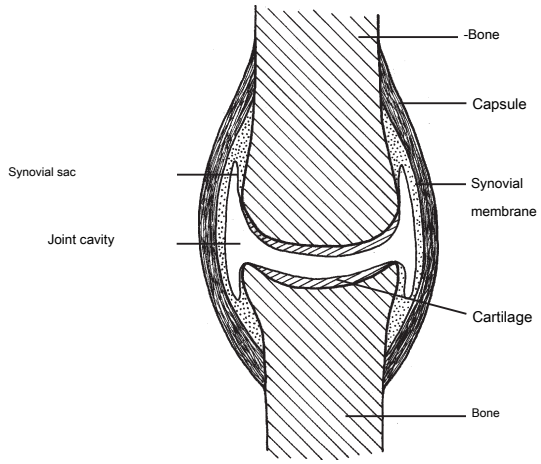


Fig. 6.8 Scheme with the locations of the main superficial and deep serous bags (B. s.). In black, those of the anterior and lateral planes. In white dots, the later ones.

The tendon is made up of hard, inelastic fibers from the end of the muscle to its bony insertion. Its color is opaque white. The tendon sheaths are sliding organs that surround the tendons in areas where their displacement is of great amplitude; They are of connective origin and similar in structure to articular synovial membranes; They are closed cavities that contain a practically virtual space.

The serous bags are located in the support and friction areas. The most superficial serous bursae or bursae are: prepatellar, olecranon, retroaquiliana, popliteal and anserine (fig. 6.8).

Fig. 6.9 Structure of a synovial joint.



JOINTS

The meeting points of two or more bone surfaces constitute the joints (fig. 6.9). This contact is made through cartilage.

The cartilage is hyaline, smooth, shiny, moist and slightly bluish; It is composed mainly of water.

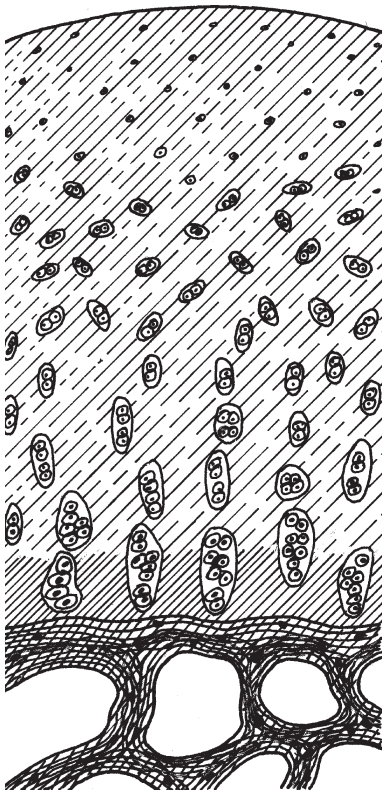
Unlike other fabrics, it is very firm and constitutes the only pressurization system.

The cells are arranged in three layers: the superficial one, whose chondrocytes are flattened; the medium, in which they adopt a larger rounded shape, and the deep, with cells separated by a calcified matrix that rests on the bone lamina (fig. 6.10).

The cells are separated by a homogeneous mass, composed of collagen fibers and protoglycans. These fibers anchored in the layer of calcified cartilage curve forming arches (fig. 6.11).

Protoglycans and type II collagen fibers provide the basis for maintaining the pressure of these tissues, which can be as large as three or more atmospheres. These aggregates are made up of chondroitinsulfate and keratinsulfate. When protoglycans are lost, as happens in different types of joint diseases, the cartilage softens and loses its resistance. Normal articular cartilage does not

Fig. 6.10 Cut of cartilage.



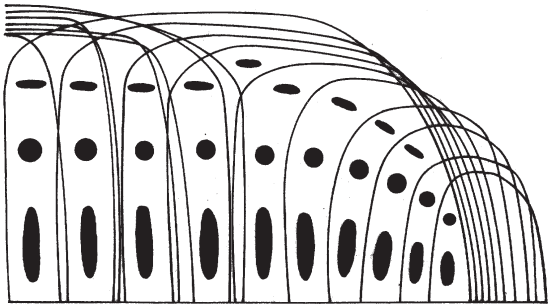


Fig. 6.11 Collagen fibers of cartilage.

It remains static, but is constantly renewing itself. Normal hyaline cartilage lacks vessels and nerves, this means that the nutritional needs of chondrocytes are met through the vasculature of adjacent tissues. In most cases it is thought that this is a function of the synovial capillaries.

Synovial fluid is the vehicle that carries nutrients to the chondrocytes and returns their metabolic wastes to the bloodstream.

The synovial membrane is among the most important joint tissues; It has a fibrous external part that constitutes the joint capsule reinforced by ligaments and a soft internal part. This free surface of the synovium is lined with more or less epithelioid fibroelastic cells. The synovium is rich in vessels and nerves.

The synovium or synovial membrane presents a characteristic pattern: a matrix composed of microfibrils and abundant protoglycan aggregates. Within this matrix lie the synovial cells; These have been classified as type A cells, similar to macrophages, and type B cells, similar to fibroblasts. The most important thing about these cells is their capacity for synthesis and protection of the synovium. The synovial cells give off fingerings without permeable intercellular junctions.
The synovial membrane is one to three layers of cells deep.

Synovial tissue is the seat of many inflammatory disorders known as synovitis.

The menisci are composed of fibrocartilage, on the one hand they adapt to the bony extremity and superficially they are inserted into the joint capsule.

synovial fluid

The joint cavity contains the synovial fluid secreted by the synovial membrane. It is rich in hyaluronate, which gives it its viscosity, similar to egg white. Hyaluronate allows articular surfaces to slide easily against each other and over exposed articular cartilage.

The synovial fluid also contains a small amount of leukocytes, less than 300 mm', mostly mononuclear cells with macrophage power; polynuclear cells represent less than 25% of the cells; Sometimes cartilage fibers are found from cartilage deterioration.

The muscles that make up the rotator cuff do not need to contract to keep the arm hanging in relaxation below the shoulder; The joint does not dislocate under the force of gravity because its components remain glued. This property is due in part to atmospheric pressure. But studies also show that there is an adhesive factor involved in this mechanism. The most plausible explanation is that the synovial fluid serves as glue.

Friction is the inevitable result when one surface slides over another. It is expressed as the coefficient of friction, a measure of energy. Many experimental studies show that the coefficient of friction is markedly low in normal joints.

It seems that the joints have a very high lubrication system. The most important component of joint lubrication is supposedly the boundary layer.

tant.

Lubricin is a specific glycoprotein produced within synovial joints and appears to be the main adhesive substance. Phospholipids also play an important role in the limiting layer.

All the mechanisms that we have mentioned contribute to maintaining joint stability:

-Atmospheric pressure.

- Synovial fluid, as an adhesive component.

- Low friction coefficient.

Classification of joints Diarthrosis

Diarthrosis or synovial joint is the most common and important type in rheumatological clinics. They are joints that are actively guided by muscles and tendons, stabilized by ligaments, covered by hyaline cartilage, and lubricated by synovial fluid.

In mechanical terms, synovial joints consist of two well-lubricated contact surfaces that allow sliding across, against, or around each surface with the other.

Synovial fluid lubricates the joints themselves (cartilage on cartilage) and the adjacent structures (synovial on cartilage and synovial on synovial). These areas of additional contact are mirrored and provide the redundant surface area required to facilitate a full range of motion at each synovial joint.

Synarthrosis

Synarthritic joints allow movement between two adjacent bones, but there is no independent movement; Its articular surface is rudimentary and tends to disappear over time.

Movement has various patterns that depend on mobility and stability requirements. These demands are unique among the adjacent flat bones of the skull, so they present a unique pattern. In synarthroses, movement has a slow range of growth rates, widening to accommodate the mature brain.

Amphiarthrosis

In this type of joint, the bones are joined by a flexible segment of fibrocartilage. Such joints are most visible in the thoracic cage, where costal cartilage joins the end of each rib to the sternum. This fabric curves easily to accommodate respiratory movements and also to provide to the heart and lungs with flexible and hard joints that protect them from thoracic trauma. Similar joints provide an element of flexibility to the pelvic girdle.

At the pubic symphysis, fibrocartilage forms a strong bond that protects bones during acute trauma. In response to the hormone relaxin, these joints normally loosen in late pregnancy, providing a wider, looser birth canal. In the posterior part of the pelvic girdle, the sacroiliac joints are a little more mobile and have a synovial pattern. Movement decreases in older subjects, of either sex, and they suffer from ankylosis.

Intervertebral discs have a special form of amphiarthrosis between adjacent vertebral bodies.

The nucleus pulposus interposed between the vertebrae provides the body's only hydraulic system, which distributes force loads widely and across the vertebral surfaces, and accumulates load energy between the fibrous rings that surround it.

PHYSICAL EXAMINATION OF THE OSTEOMIOARTICULAR SYSTEM (SOMA)

General guidelines

A thorough examination of SOMA is a lengthy procedure, including evaluation of skeletal muscle strength and measurements of limbs and joint mobility.

This type of examination may not be indicated for all people, or may not be tolerated, due to fatigue or prolonged exertion during activity. If a subject does not show striking signs of osteomyoarticular dysfunction, a less in-depth screening examination may be sufficient.

To determine if a screening exam is sufficient, ask the subject the following questions: Do you have any pain or swelling in any limbs or when you move? Does this pain affect your daily activities? Do you have a history of injuries to any muscles, bones or joints? Additionally, note any apparent deformities during the general physical examination inspection, such as gait, standing, or inappropriate body alignment.

In order to rule out alterations in SOMA, observe the subject's way of walking, how he moves from a sitting to a standing position, how he clenches his hands or manipulates his clothes.

If these simple techniques do not indicate osteomyoarticular problems, the following components can be eliminated from the examination: careful examination of the muscular strength of each muscle, detailed range of movements, and measurements.

Even if the subject needs a thorough examination of the SOMA, he or she must judge whether certain procedures or maneuvers are appropriate. In trauma cases with suspected bone fractures, for example, range-of-motion nanoparticles could cause considerable pain and aggravate the injury. In general, if superficial or deep palpation of the structures causes pain and the cause of the pain is uncertain, joint mobility should not be explored until its cause can be known.

Likewise, if pain appears during mobility maneuvers, take special care not to cause further injury. In the case of an extremity injury, you should first examine the unaffected side to determine the person's usual osteomyoarticular function.

Neck and spinal movements are strictly contraindicated if the person has had an accident that may have caused spinal injury.

Exposure. The examined subject may remain clothed, in the examination gown, or covered, during most of the osteomyoarticular examination, especially when examining the distal parts of the extremities. You should completely undress when examining body alignment and spinal configuration.

GUIDE TO EXPLORATION AND THE WRITTEN RECORD

You should focus the exploration of the SOMA and its written record on the following aspects:

- Structural symmetry and alignment.
- Ease and range of movement.
- Muscle mass and tone.
- Muscular strength.
- Appearance of the skin over the joints.
- Pain, crepitation and deformities.

Exploration of SOMA is carried out through inspection, palpation and measurements. Percussion and auscultation are not routine examination methods in the entire SOMA examination; They are performed in certain areas, to reveal pathological osteomyoarticular alterations or other systems.

The muscles should also be examined, as part of the neurological examination (coordination of movements, muscle strength, etc.), since the muscle examination also includes the evaluation of neurological function.

MEASUREMENTS

In the complete SOMA examination, measurements are useful to describe the length and circumference of the arms and legs and the range of motion of the joints.

Take limb measurements with a cloth or other flexible material tape measure if there appears to be any unevenness between the right and left sides.

The limits for measurement of extremities may be designated as follows:

1. Complete upper limb. From the greater tuberosity of the humerus to the styloid process of the radius (some describe it from the acromial process to the tip of the second finger):

   a) Arm: from the acromial process, or the tuberosity of the humerus, to the olecranon process.

   b) Forearm: from the olecranon to the ulnar styloid process.

2. Complete lower limb: From the anterior superior iliac spine, or from the greater trochanter, to the tibial malleolus:

   a) Thigh: from the anterior superior iliac spine, or the greater trochanter, to the middle condyle of the knee.

   b) Leg: from the middle condyle of the knee to the tibial malleolus.

The measurements in length and circumference of the compared structures must be equal. A centimeter difference between the right and left extremities is considered clinically insignificant.

The arc or range of joint movement must be measured in degrees, with a portable device called a goniometer. The zero reference (0) on the arm of this instrument must be aligned with the neutral position of the joint (anatomical extended position). The person must move the joint through a specific range of motion and maintain the final position, while the other arm of the goniometer is moved to this position and the angle is measured. This measurement can be compared with normal values. Record only values that deviate 10-20% from normal. Small differences in range of motion may be noted when comparing joint motion between the right and left sides.

SOMA EXPLORATION

EVALUATION OF RUDE MOVEMENTS AND POSTURE

As expressed in the general physical examination chapter, observe the person's stay and movements, from the moment they enter the room, while they are standing and sitting, when they gesture and when they manipulate their clothes. Next, ask him to stand and walk, to detail the gait, posture, stay, and range of motion of the joints used for walking.

Normal gait and good posture or attitude were already described in the aforementioned chapter.

EXPLORATION OF SOMA BY STRUCTURES

As the SOMA is made up of bones, muscles and joints, we will first give a general outline of the elements to take into account in the separate exploration of each of these structures.

Physical examination of bones

Inspection. The inspection should be carried out by comparing the bone symmetry, between the right and left sides. The existence or not of bone deformities must be sought,

swelling or edema of the soft tissues and changes in skin color.

Palpation. The existence or not of provoked pain must be explored, and the absence of deformities, diffuse or localized volume increase, volume decrease or bone depressions must also be confirmed. Bone mobility should also be explored in limb segments where there is no joint, to rule out abnormal mobility or crepitation.

Measurement. Measurement of the limbs is an important part of a more detailed physical examination. It is necessary to carefully measure the axial lines of the extremities. With them we will observe whether or not there is a difference in the length of the limbs.

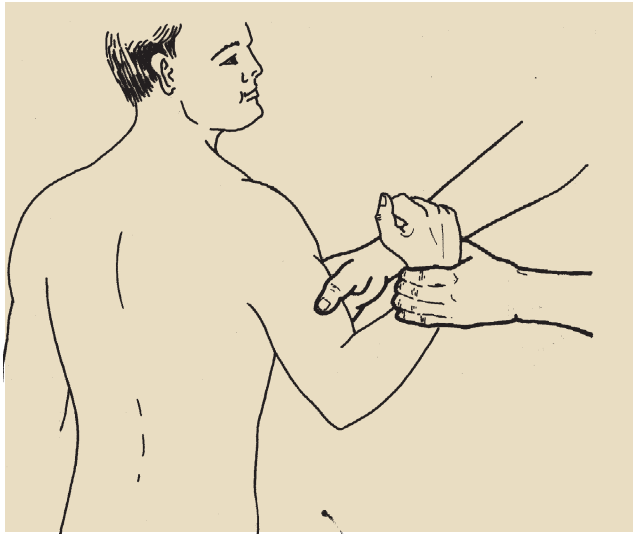
Physical examination of the muscles

Inspection. It should be done with the patient naked, comparing one side with the other. We will explore muscle volume, whether or not there are atrophies or tumors. We will also take into account the inspection, the active movements of the muscles.

Palpation. We explore whether or not there is pain on palpation, the consistency of the muscle, passive mobility and muscle strength opposing the effector movement (fig. 7. 1).

Measurements. Muscle contour measurements will also be performed, if necessary. To do this, we take a bone reference point and an equal distance from it on both extremities, we determine its circumference, and thus we will see if it is increased or decreased. Serial measurements of limb circumference are most valuable when boundaries or marks made on the person's skin are specified, with marking.

Fig. 7.1 Muscle examination: biceps.



non-toxic res, to indicate the place of placement of the centimeter.

physical examination of the joints

The joint physical examination is also performed in a comparative manner (both hemibodies). The systematization can be: from the upper joints to the lower ones, and the spine, which can be explored at the beginning or at the end.

For each joint, the following sequence must be followed: inspection, palpation and mobility (range of motion or ranges of joint mobility), which may or may not include measurements with the tape measure or goniometer.

Due to its importance, it will be analyzed in detail in the exploration of SOMA by region.

When palpation is to be performed, ask the subject to express whether there is pain or any other type of sensation.

During joint mobility maneuvers, keep the palm of your hand over the person's joint to detect crepitus (cracks) and deformities.

Evaluation and recording of joint mobility

Every joint in the body has a normal range of motion, or maximum possible motion.
Joint movements are described by the measurement in degrees of the arc of amplitude and by the type of movement. When joint mobility is stated in degrees, the neutral joint position is zero degrees. An example of the description of the movement of a hinge joint such as the knee is: flexion, 130°. Movement may be possible in several directions, depending on the type of joint.

Differences in exercise, mental health, and genetic levels lead to normal differences between people. Generally, joint movement is performed easily if the degree of movement differs between 10 to 20% of the maximum possibility.

The degree of movement is normal if it occurs without stiffness, pain or crepitus. Joint movement beyond the maximum possible may be abnormal, indicating ligament tears, abnormal connective tissue, or joint fracture.

SOMA EXPLORATION BY REGION

After evaluating gross movement and posture, the examination of the osteomyoarticular system (SOMA) focuses primarily on the examination of the extremities and spine.

The examination of SOMA in other parts of the body is integrated with the examination of each region. For example, in The examination of the head explores the tempo-

romaxillary: during the chest examination, the muscles, bones and joints related to pulmonary ventilation are evaluated, and in the genitourinary and rectal examination, the muscles related to intestinal and bladder evacuation are evaluated.

Vertebral column, paravertebral muscles, scapulae V pelvis

Anatomophysiological count of the spinal column

The vertebral column is shaped like a long bony stalk, located at the back of the trunk, in the midline.
Protects the spinal cord by wrapping it; It is the skeletal element that supports the head, shoulder girdle and upper limbs and transmits its weight to the pelvis and lower limbs. It is made up of vertebrae joined together by intervertebral discs and ligaments. It contains within it the spinal cord and its roots, and the meningeal covers.

The spinal column is divided into four segments:
the cervical region (fig. 7.2) with seven vertebrae, the dorsal with twelve, the lumbar with five and the sacrococcygeal with nine or ten.

The vertebrae are the bony elements of the column, with an anterior and posterior arch, joined by the pedicles; The anterior arch is the vertebral body itself and the posterior arch consists of the spinous processes, the transverse processes and the articular facets. Between both arches, anterior and posterior, is the spinal canal.
The vertebrae have special characteristics in each of their segments. They have semiological interest only from the imaging point of view.

The vertebral bodies are articulated in their anterior part by the intervertebral discs and in their posterior part, by the interapophyseal joints. The intervertebral disc is of the amphiarthrosis type. From a functional point of view, the disc has the role of cushioning pressure, while the nucleus pulposus constitutes the place through which the axis of the movements of the discosomatic joint passes. The disc is a very delicate vertebral structure and receives mechanical punishment, which is why its involvement is the most frequent among all the components of the spine.

The intervertebral disc consists of: the annulus fibrosus, the nucleus pulposus and the cartilaginous plates. The nucleus pulposus has a high water content (88% in the newborn) that decreases throughout life (70% or less in the elderly). Normally, the standing individual supports pressures produced by the supra- and underlying elements of the spine, which are subsequently distributed among neighboring structures. When it loses water, as a result of age or injuries to the annulus fibrosis, it loses its elasticity and capacity for expansion.

There is no intervertebral disc between the atlas and the axis.
In addition to the fibrous disc annulus, which constitutes a powerful means of union between two neighboring vertebrae, there is a series of ligaments in the spine that are important in spinal conditions.

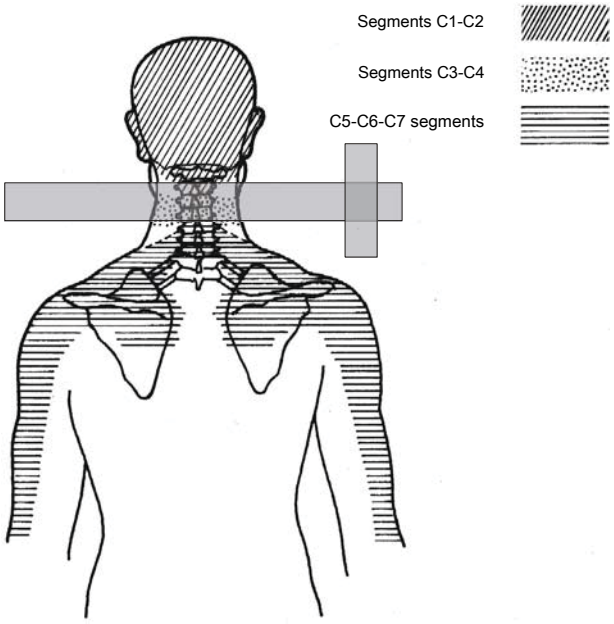
Inspection

The subject is placed standing, with his arms hanging along his body, preferably naked. In the case of frontal and profile examination, especially in women, the use of essential underwear could be permitted, which allows the visualization of the structures, while respecting modesty and avoiding an embarrassing situation for the subject; In the back examination, which must be performed with the subject naked, an examination gown open at the back could be used as the only clothing, if available. The lighting must be adequate and coincide (behind the examiner) with the area being explored.

1. Ask the person to stand facing forward, at attention, with heels together and toes slightly apart, and note the alignment and symmetry of the shoulders, pelvis, and knees.
   Remember that the shoulders, iliac crests and knees should be aligned at the same level on both sides.

   The bones and muscles on each side are symmetrical, with respect to size, shape, and function. Surface reliefs such as trochanters, crests, spines and other bony prominences should also be symmetrical.

Fig. 7.2 Cervical segment of the vertebral column.



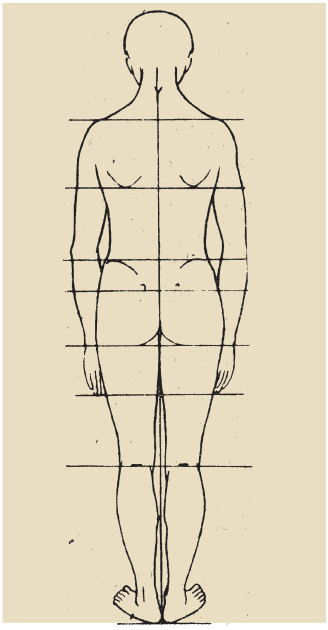
2. Look at the person in profile and observe the alignment and whether the spinal curvatures are normal or if there is exaggeration or rectification of any of them.
Despite having described the characteristics of normal body alignment in the "General physical examination" chapter, it is worth remembering again that in a lateral view there is a normal alignment if an imaginary vertical line drawn passes through the earlobe, the shoulder, the hip, the femoral trochanter, the center of the knee and in front of the ankle. The elbow, finger, ankle and knee joints should be slightly flexed.

Normal curvatures of the spine, profile view, with the subject standing. When the spine is viewed from the side it is characterized by a concave curvature in the cervical spine, convex in the dorsal and again concave in the lumbar, the transitional points being harmonious.

Abnormal curvatures of the spine, profile view, with the subject standing. Exaggerated curvatures should be considered abnormal, even when oriented in a normal direction. Lordosis is the abnormal concavity of the lumbar spine; Kyphosis is the abnormal increase in dorsal curvature. Hump is a posterior projection of the spinal column (see more details in the Propedeutics Section).

3. Ask the person to stand on their back, in the same "at attention" position, and observe the symmetry of the spine, shoulders, scapulae, iliac crests, gluteal folds and knees (fig. 7.3).

Fig. 7.3 Normal subject.



When looking from the back, the spine should be straight.

The pelvis should be well aligned horizontally; Otherwise, other maneuvers are used that will be described in the Propedeutics Section.

4. Ask the person to bend forward at the waist, first on their back, then in profile. Observe the ease of movement, the orientation of the spine and scapulae, and the curvatures of the spine.
From behind, the entire spine should appear convex and regular, the vertebrae should remain in the midline and the scapulae at the same height, on the same horizontal line.

In profile, with the subject leaning forward, the entire spine is seen as a regular curve.

Mobility exploration

1. Ask the person to bend forward at the waist to assess spinal flexion. Measure the arc of motion by determining the width of the angle between the neutral and flexed positions (normal flexion: 90°) or by measuring the length between the tips of the fingers and the floor.

An alternative method is to measure the change in length along the spine while the person leans forward. To do this, place the measuring tape from the spinous process of C7 to the spinous process of S1. Keep your hands within these limits, but allow the tape to slide through your fingers at S1, as the person leans forward. Normally the length should increase around 10 cm in adults. If the length does not increase, conditions that limit vertebral joint mobility should be suspected as a cause of the spine remaining rigid with forward flexion.

2. Explore vertebral extension by asking the person to lean back. Normal extension: 30°.
3. To explore the lateral movements of the spine, ask the person to lean laterally, as if they were going to touch the side of the knee, on one side and the other.

Normal lateral movements: 50°.

4. Next explore spinal rotation, as the person rotates the head and shoulders as a unit, first to the left and then to the right, while keeping the pelvis stationary.

Palpation

1. Palpate the muscles of the shoulder and pelvic girdles, after inspection, to rule out pain, swelling or muscle atrophy.

2. Palpate the spine with your fingertips.

It can be done with the person sitting, standing or in a prone position. Note if there is any increase in volume, pain, or bone deformities.

The spinous processes should be compressed and displaced laterally, taking them between the index fingers and thumb, looking for pain. Subsequently, we apply compression to the points located between two spinous processes, 2 cm on both sides of the midline (emergence of the nerve roots), checking whether or not there is pain. Then, the paraspinal muscles are palpated to rule out muscle spasticity and lightly tapped along the entire length of the spine, with the ulnar surface of your hand, with a finger or with the percussive hammer, to rule out the existence of any pain. Finally, passive movements of the spine are performed, segment by segment.

Muscle strength assessment

Extensor muscle strength is best assessed with the person in the prone position. Instruct the person to try to lift the head and shoulders while you apply resistance by placing your hands between the shoulder blades.

Ask the person to repeat the spinal rotation movements while you place your hands against the left and right shoulders, respectively.

Special maneuvers

Some of the special maneuvers will be described when studying each segment, in this Section.

Because all the special maneuvers are used to demonstrate the existence of organic disease, and because they all show greater positivity if there is an inflammatory component, most of them will be described in the Propedeutics Section.

You can postpone any maneuvers that require the person to lie down until the evaluation of the lower extremities.

Cervical spine

Inspection

It will be performed with the subject sitting, to look for deformities and evaluate their active mobility. The person is asked to perform extension, flexion, lateralization and rotation movements of the spine, to explore the limitation of those or the provocation of pain.

The subject should also be asked to rotate the neck on the vertical axis of the body, first to the right and then to the left or vice versa, in search of limitation, pain or "dizziness."

Mobility exploration techniques

1. Explore flexion and extension of the cervical spine by asking the person to tuck their chin to their chest and then bring their head back.

During flexion, the chin should normally touch the sternal notch; If it is not reached, we can speak of limitation; In extension, the separation between the chin and the sternal fork must reach a minimum of 18 cm.

2. To explore lateral sway of the cervical spine, ask the subject to try to put the ear to the shoulder, while keeping the shoulders still.
3. Explore rotation of the cervical spine by asking the person to turn the head toward the right and left shoulders, respectively, while keeping the shoulders still.

Evaluation of the ranges of mobility

side view	Neutral line: 0° (vertical passing through the ear and shoulder) Flexion: 45° Extension: 550
Front view	Normal lateral sway: 40° on each side of the midline Normal rotation 70° to each side of the midline

Palpation

Compression (supplemented by percussion) and mobilization of the spinous processes must be performed. When compressing between two spinous processes (1.5 cm outside the midline), the presence of pain indicates irritation of the explored root. Passive mobilization of the head will be carried out in an anterior, posterior and lateral direction, to demonstrate limitation of movement or the existence of provoked pain. Active rotation movements must be performed; normal up to 60° without causing pain.

The joint mobilization-palpation maneuver allows us to check whether crepitation exists or not.

Compression of the vertex of the skull in a vertical direction is also performed, which normally should not cause pain (fig. 7.4).

Exploration of muscle strength

Test muscle strength by asking the person to repeat the flexion and extension movements, while pressing your hand against the forehead during flexion and against the occiput during extension.

To assess muscle strength during lateral swing, apply pressure to the right occipital region and

Fig. 7.4 Skull compression maneuver.

left, respectively, opposing the movements made again.

Finally, apply hand resistance to the temporalis and ask the person to repeat the rotation movements.

Dorsal column

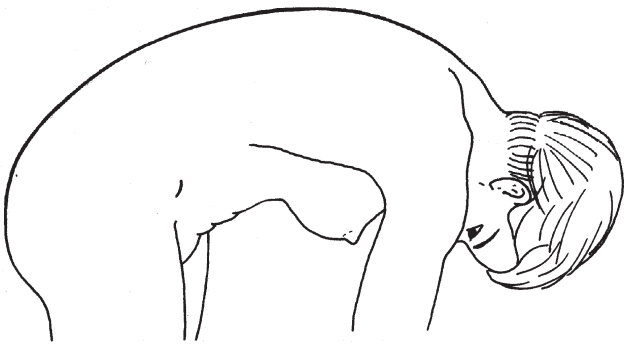
Inspection

As mentioned above, the normal dorsal column, seen with the person standing, in profile, has a slight convex curvature, and seen from the back, it should be straight, or almost straight, with a slight curve secondary to the process of unequal development of the shoulder girdle in right- and left-handed people.

Scoliosis is a lateral deviation of the dorsal spine, seen from the back. It is corroborated by using a plumb line held by a thread, which is fixed to the spinous process of the seventh cervical vertebra, with the patient standing. This, in addition to demonstrating scoliosis, serves to check whether it is compensated or not. If so, the thread passes through the intergluteal fold.

If scoliosis exists, other maneuvers must be performed that will be described in the Propedeutics Section. We will only add that if the scoliosis disappears when exploring the

Fig. 7.5 Trunk flexion: normal rounded curvature.



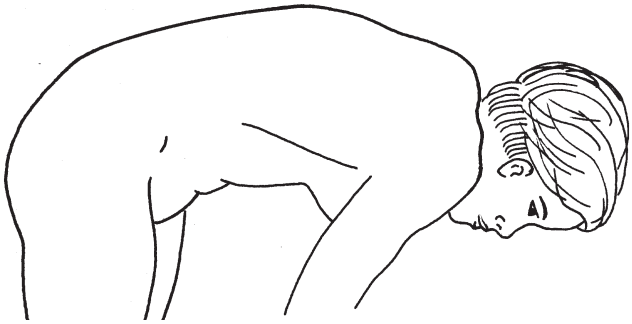


Fig. 7.6 Exploration of the spine: rectification of the lumbar segment.

seated subject, it is due to the shortening of a lower limb.

Palpation and percussion are combined to look for pain, using the Finck maneuver (see Section II).

Measurements

Measurements of the lower limbs should be performed, taking as reference points those described above when there is scoliosis and it is suspected that it is due to shortening of a limb.

Thoracic perimetry should be performed with a measuring tape (in inspiration and expiration), in order to explore the costovertebral and costotransparent joints.

VersaS.

lumbosacral spine

Inspection

The lumbosacral spine is examined with the person standing with their back to the light.

It should be observed with the subject leaning forward and legs extended, in a lateral position with respect to the explorer, to check whether a rectification of the lumbar segment occurs instead of the normal harmonic curve of said segment (figs. 7.5 and 7.6).

It is also observed from behind, in the erect position, if both gluteal folds are at the same level or there is descent of one of them, which is pathological.

Palpation

It is performed by mobilizing the spinous processes between the index fingers and thumb, as already described. Palpation of the paraspinal muscles will also be performed. If contracture is detected, other maneuvers will be performed that will be described in the Propedeutics Section. The rest of the palpation is the same as throughout the spine.

Special maneuvers of the lumbosacral spine will also be described in the Propedeutics Section, although they must be known to explore a supposedly healthy individual and rule out lumbosacral affection.

sacroiliac joint

Anatomophysiological count

The sacroiliac joint constitutes a diarthrosis.

During childhood and youth it has a joint cavity and a synovial membrane that guarantees certain mobility. Evolutionarily, adhesions appear between the articular cartilages, which fuse; The joint cavity disappears completely and the joint becomes an amphiarthrosis, without any mobility.

The articular surface that corresponds to the iliac bone is called the atrium. The articular facet of the sacrum is located in the upper half of its lateral surface, at the level of the first two sacral vertebrae. In adults, the articular facets present marked irregularities, reversed in both bones, so that when they articulate they fit together. The two bones that form the joint are joined by a joint capsule and ligaments; the fibrous capsule is very short; The anterior sacroiliac ligament, which joins the two bones in front, tears easily.

The posterior sacroiliac ligament is very powerful. There is a synovial membrane, but usually there is no synovial fluid inside, except in pregnant women.

This joint lacks its own muscles; The gluteus maximus is responsible for straightening the coxalis over the femur and the spinal muscles are responsible for extending the vertebral column over the sacrum.

The sacroiliac joint is always mobile in children and retains some mobility up to age 40 in men and up to age 50 in women. The disappearance of joint movement in adults is explained because the sacroiliac joint suffers early degenerative alterations.

These movements are passive, due to the pressures and counterpressures suffered by the sacrum and iliac bones, so the ligaments play the main role, instead of the muscles, in the movements of this joint.

Examination of the sacroiliac joint

Inspection

The arrangement of its articular facets means that the sacroiliac joint is out of reach of any exploration from the back, since the iliac tuberosity covers it completely, leaving a single point accessible, which is the posteroinferior end of the articular facet, a finger's width below the posterosuperior iliac spine, at the level of the second sacral foramen, described by Rotés-Querol, together with Forestier and Jacqueline.

The inspection provides very little data, even when there are alterations. Mobility, at the ages in which it is present, is scarce, so its exploration has no value.

Palpation

Sometimes inflammatory changes are noted. Pain on pressure in the point indicated as the only one open to exploration.

The special maneuvers of the sacroiliac joint are to detect pain, when its involvement is suspected and, although they must be known from now on, they will be described in the Propedeutics Section (see Chapter 30).

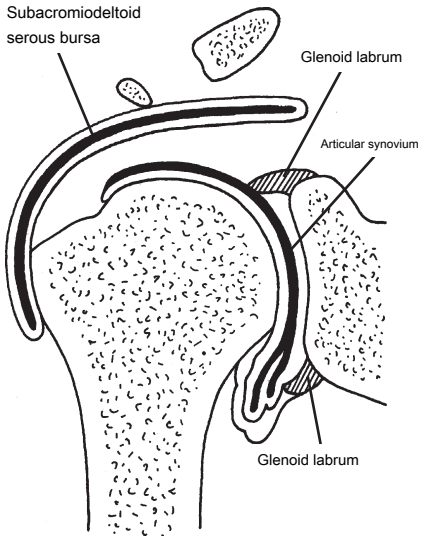
Shoulder joint

anatomical count

This joint is made up of the shoulder blade or scapula, the clavicle and the upper end of the humerus (fig. 7.7).

The scapula is a flat, triangular bone that applies to the posterior surface of the thorax. The posterior surface of the scapula is divided into: supraspinous fossa, which occupies the upper 1/4 and the infraspinous fossa, which occupies the lower 3/4, separated by a transverse eminence, the spine of the scapula. The outer end of the spine constitutes the acromion; On the inner edge of the acromion there is an articular facet destined for the outer end of the clavicle, it is the aChromioclavicular joint. On the upper edge of the scapula and near its external angle, there is an eminence, the coracoid process. On the outer edge of the scapula there is an oval surface, the glenoid cavity, intended to articulate with the humeral head.

Fig. 7.7 Diagram of a cross section of the scapulohumeral joint showing the arrangement of the synovium and the subacromiodeltoid serous bursa. Note the inferior fold of the capsulosynovial formation that distends during the abduction movement: when there are adhesions at this level, movement is limited (according to modified Testut).



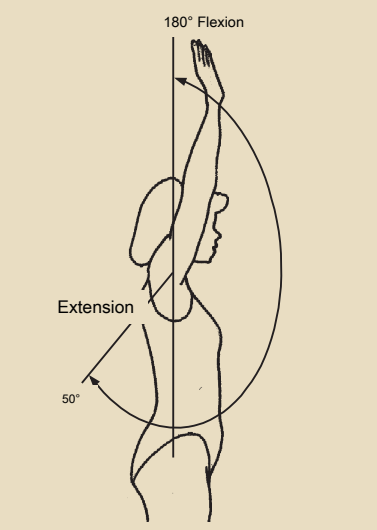


Fig. 7.8 Shoulder flexion and extension.

The clavicle is a long bone that articulates with the acromion on its external end and with the sternum on the internal part.

The humeral head articulates with the glenoid cavity;

Next to the anatomical neck of the humerus we find a voluminous eminence (trochiter) and, inside it, another minor eminence, the trochin. Between the two is the bicipital slide, through which the tendon of the long head of the biceps slides.

A. Scapulohumeral joint.

It is formed by the humeral head and the glenoid cavity. A fibrocartilage (glenoid labrum) adheres around the entire contour of the glenoid cavity, increasing the articular surface. This joint is endowed with powerful musculotendinous formations that play a decisive role in its function and pathology. In this joint everything is organized according to movement, in contrast to the joints of the lower limbs, whose anatomy adapts to support.

B. Acromioclavicular joint.

It is detected by sliding the finger along the upper surface of the clavicle, towards its distal end. Collaborate with shoulder movements.

C. Sternoclavicular joint.

Made up of the clavicle and the sternum. Between both articular surfaces there is a meniscus that, due to its arrangement, prevents the displacement of the inner end of the clavicle inward.

Clinical examination

Inspection

Inspect the shoulders from the front, with the person standing, sitting or lying down, and note the symmetry of the sides.

two right and left. Inspect the skin over the collarbones for lumps. Observe and note the posture of the shoulders (erect, slumped, hunched).

Mobility

The shoulder movements to explore are: flexion, extension, abduction, adduction, external rotation and internal

The arcs of normal mobility, measured in degrees,

Are:

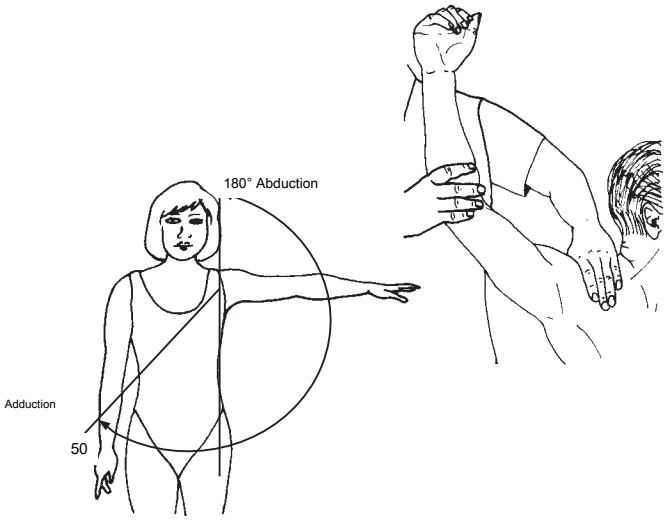
- Flexion: 180°
- Extension: 50°
- Abduction: 180
- Adduction: 50°
- External rotation: 90°
- Internal rotation: 90°

To explore the flexion, ask the person to raise their arms in front of them, until they are pointing above their head. To explore extension, ask him to extend his arms back and up from his resting position (arms down, fingers pointing toward the floor) (Fig. 7.8).

In flexion, the lower end of the humerus is directed forward and upward, the arm reaches 120° and can then be raised up to 180°. In extension, the movement can reach up to 90°, but from 30° onwards, the movement is carried out in the scapulothoracic joint.

For abduction, instruct the person to spread the arms apart laterally until the fingers point above the head (Fig. 7.9 a). If the patient is instructed to separate the arm from the trunk,

Fig. 7.9 Shoulder abduction: a, active; b, passive, holding the scapula.



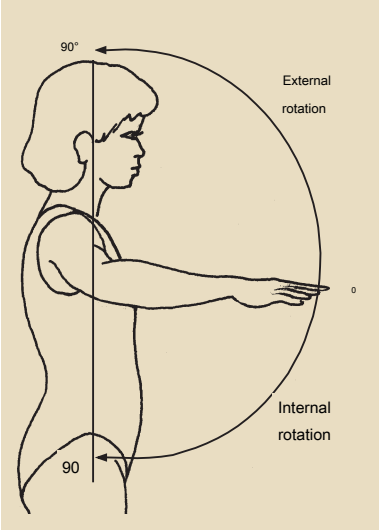


Fig. 7.10 Shoulder external rotation.

Holding the clavicle and scapula with the thumb, index finger, and scapula prevents involvement of the scapulothoracic and sternoclavicular joints (Fig. 7.9 b). If shoulder fixation is complete, abduction does not exceed 110° or 120°, instead of reaching 180°.

Test adduction by asking him to bring his hand to his chest. Finally, to explore the internal and external rotations of the shoulder joint, ask the subject to raise the arm in front, to the level of the shoulder and flex the elbow, placing it at a right angle, keeping the hand with the fingers extended, pointing away, and the palm facing the ground.

Observe the external rotation of the shoulder as the subject moves the forearm upward and outward (Fig. 7. 10).

Assess internal rotation as the subject rotates the forearm inward and downward so that the fingers that were pointing up at the end of external rotation now point toward the floor at the end of internal rotation. Internal rotation (Fig. 7.11) best explores the acromioclavicular and scapulohumeral joints if performed by asking the subject to reach the spine with the back of the hand. It can reach the D6 vertebral body. If movement is limited, reach D7-D8-D9.

Palpation

With your fingertips, feel along the clavicle from the inside out, towards the shoulder, and check for any discomfort or deformity.

Locate the sternoclavicular and acromioclavicular joints, at their inner and outer ends, respectively, as you move your fingers across the collarbones.

Locate the greater tuberosity of the humerus by palpating the shoulder while the subject abducts and adducts the shoulder, allowing you to differentiate the glenohumeral joint, between the scapula and the humerus.

Ask the subject to externally rotate the shoulder and palpate just medial to the greater tuberosity (trochyterus) to locate the long head of the biceps tendon. Palpate along the tendon (which looks like a cord) for pain. Then palpate the deltoid muscle.

Explore passive mobility, if you need to rule out limitation of movements or specify pain in them and, finally, evaluate muscle strength.

Muscle strength assessment

Apply resistance to the anterior portion of the arm above the elbow and ask him to perform the flexion again.
Then apply pressure to the posterior surface above the elbow and explore muscle strength during shoulder extension.

To test muscle strength during abduction, apply resistance by placing your hand on the outside of the arm above the elbow.

To explore muscle strength during adduction.
Apply resistance to the medial portion of the arm above the elbow.

elbow joint

anatomical count

The humeroulnar, humeroradial and superior radioulnar joints form the elbow joint, in which bone formations are found such as: lower extremity of the humerus, upper extremity of the ulna and upper extremity of the radius.

At the lower extremity of the humerus, two lateral eminences stand out, the epicondyle, on the external side and the epitrochlea, on the internal side. Between the two is the articular surface made up of the condyle on the outside and the trochlea on the inside (fig. 7.12).

Fig. 7.11 Internal rotation of the shoulder.

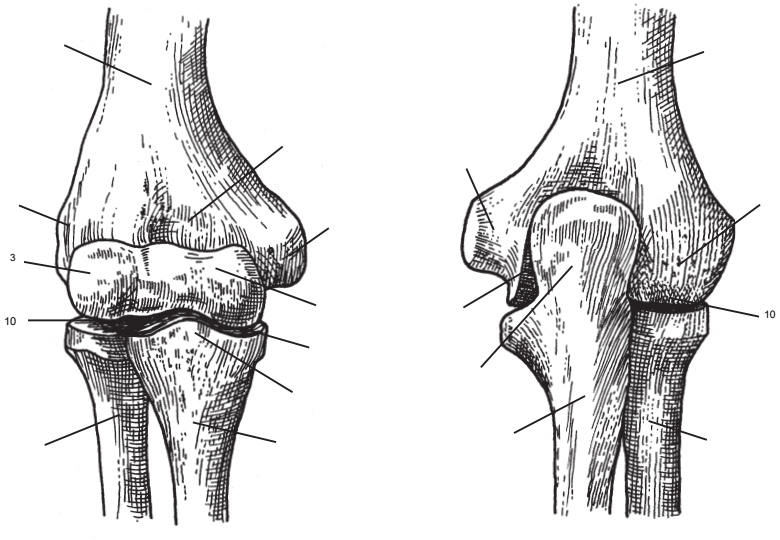


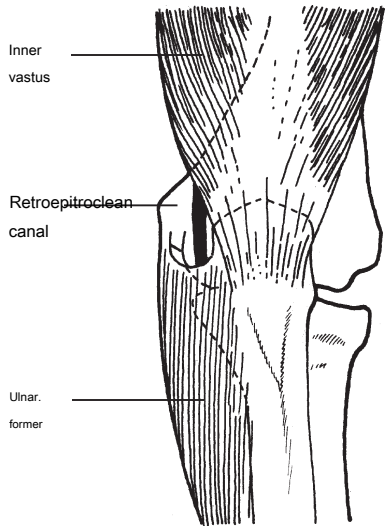
Fig. 7.12 Bone formations of the elbow joint seen from its anterior and posterior aspects, respectively: a, humerus;
b, ulna; c, radius;1, epicondyle; 2, epitrochlea;
3, condyle; 4, trochlea; 5, greater coronoid fossa; 6, olecranon fossa; 7, olecranon;
8, greater sigmoid cavity; 9, coronoid process; 10, glenoid cavity.

The upper part of the ulna articulates with the humeral trochlea, where there is a large articular cavity, the greater sigmoid cavity; It is limited behind by a large process, the olecranon, and below and in front, the coronoid process.

The lesser sigmoid cavity, on the external surface, articulates with the radial capsule. On the upper surface of the radius there is a depression or glenoid cavity, which adapts to the humeral condyle. The musculotendinous formations of the elbow joint (fig. 7.13) are made up of the biceps, whose tendon inserts into the radius, and the brachialis anterior, which inserts into the ulna.

The proximal part of the forearm muscles that move the hand is made up of several muscles that make up the back of the forearm. In the posterior part of the joint there are almost no muscles, the bony reliefs can be palpated.

Fig. 7.13 Musculotendinous formations of the elbow joint.



The most important serous bag that we find in this joint is the olecranon bursa, a frequent location of gout tophi.

Clinical examination

Inspection

The elbow normally presents a certain degree of abduction and forms an angle open outwards of about 170°.

Inspect the existence or not of any deformity carefully. Compare the opposite sides.

Mobility

In the elbow there are two groups of movements: flexion-extension and pronation-supination. In flexion, the forearm and forearm form an angle of 30°. In extreme extension, the arm and forearm form an angle of 180°.

Not only the elbow is involved in prono-supination movements. The total extension of the rotary movement reaches 180°. It is examined with the elbow semi-flexed and reaching the hand as far as possible, in both positions.

In lax individuals, a slight hyperextension of up to 190° is achieved; In farmers or individuals who have worked repeatedly with their arms, it is rare for the extension to be complete, with 5° or 10° missing to reach 180°.

To explore flexion-extension, instruct the person to keep the arm straight while bending the elbow in a way that allows the fingers to touch the shoulder.

The opposite of this movement is extension.

Explore supination and pronation. Ask the person to extend their forearm or rest it on a flat surface, palm down. Supination occurs when the person rotates the forearm so that the palm of the hand faces upward, and pronation occurs.

when the forearm rotates and the palm of the hand faces down.

After exploring active mobility, we will perform the passive movements of flexion, pronation and supination, sometimes offering some resistance to see if there is pain. The inability to extend the elbow is always abnormal.

Palpation

We will perform palpation at the level of the epicondyle, olecranon and at the insertions of the extensor muscles, to see whether or not there is pain or palpable mass.

Palpate the posterior surface of the elbow, with the index finger and thumb, while the person bends the elbow at an angle of flexion greater than 90°, while you support the forearm with your other hand.

Palpate the medial and lateral condyles of the humerus and the olecranon of the ulna. The olecranon bursa lies between the condyles of the humerus and is not normally palpable, but may be tender if inflamed. The ulnar (ulnar) nerve is palpated posteriorly, between the olecranon and the medial epicondyle.

Exploration of muscle strength

To test muscle strength during flexion-extension, ask the person to repeat these movements and apply their hand to the medial and then dorsal surface of the wrist during flexion and extension, respectively.

To test muscle strength during supination, apply resistance to the dorsal surface of the person's hand just distal to the wrist, and during pronation, apply resistance against the volar (palmar) surface of the person's thumb.

In addition to evaluating the range of motion and muscle strength of the elbow, palpate the brachial pulse on the opposite side of the olecranon and check the biceps and triceps reflexes.

Wrist joint Anatomical count

The distal end of the ulna and radius, the carpal bones, and the proximal ends of the metacarpals constitute the wrist.

At the lower end of the radius is the styloid process, on the outside, and the sigmoid cavity, on the inside, in close contact with the ulna, which forms the lower radioulnar joint. The ulna is separated from the carpus by the triangular ligament.

The carpal bones are arranged in two transverse rows; The upper one is composed, from the outside in, of the scaphoid, semilunar, pyramidal and pisiform; and the lower one, composed of trapezius, trapezoid, large bone and hamate.

In the wrist the following joints can be distinguished: inferior radioulnar, radiocarpal, midcarpal and carpometacarpal.

Due to its importance in hand diseases, we will emphasize the carpal tunnel, from an anatomical point of view. The floor presents the carpal canal and the roof is made up of the anterior annular ligament of the carpal; It extends between both edges of the carpus. The tendons of the superficial and deep flexor muscles of the fingers and the median nerve pass through the tunnel.

Clinical examination

Inspection

Grasp the person's wrist with both hands, so that both thumbs are on the dorsal surface of the wrist. Identify the bony processes of the radius (on the thumb side) and ulna (ulna).

Mobility

To test wrist flexion (palmar flexion) and extension (dorsiflexion), ask the person to bend the hand down and up, with fingers extended, respectively.

To explore radial wrist motion, ask the person to keep the elbow in line with the wrist and tilt the wrist toward the thumb side.

To explore ulnar movement, with the elbow and wrist in the same position, instruct the person to tilt the wrist toward the ulnar side.

Palpation

We will palpate the periarticular soft tissues, the joint interline, and with the patient's closed fist enveloping the thumb, we will palpate the radiocarpal joint, a slight bulge just distal to the styloid process of the radius.

We will then continue with the exploration of passive movements of the wrist, extension, flexion, laterality and rotation.

Muscle strength assessment

To test muscle strength, place your hand against the volar surface of the subject's hand during flexion and against the dorsal surface during extension. Also, apply resistance by pressing against the person's thumb to explore muscle strength during radial movement and on the side and along the little finger to explore it during ulnar movement.

In addition to exploring the osteomyoarticular structures of the wrist, take the opportunity to explore the supinator reflex (brachioradialis) and palpate the radial pulse.

Joints of the hand and fingers

Anatomophysiological count

The bony framework of the hand is made up of the metacarpals and the fingers of the phalanges. The metacarpals, five in total, articulate proximally with the bones of the second row of the carpus and distally, with the first phalanx of the fingers. They are of the condylar type, with a distal condyle and a glenoid cavity, expanded by proximal fibrocartilage. There are two interphalangeal joints in each finger, except in the thumb.

Muscles are classified into:

a) Those that move the thumb, on the outside. They constitute the thenar eminence.
b) In the internal part, the muscles for the little finger are grouped, forming the hypothenar eminence.
c) In the central portion the lumbricoid and interosseous muscles are located.

Clinical examination

Inspection

Observe the skin of the hands and the musculoskeletal reliefs, of both hands and in a comparative manner, to rule out changes in color and the absence or absence of deformities, swelling or nodulations.

The exploration of the structures of the hand focuses fundamentally on the joints of the fingers, which are susceptible to diseases that cause deformities.

Mobility

Flexion-extension and abduction-adduction of all fingers, and opposition of the thumb, are explored.

The abduction-adduction movements of the thumb are explored with a different technique than the rest of the fingers. To test flexion of the thumb and the rest of the fingers, ask the subject to open and close the hand (close to make a fist and then extend the fingers).

Explore abduction by asking the person to spread the fingers and adduction, instructing them to join them tightly together again.

To explore thumb abduction and adduction. Ask him to move his thumb away from his hand and then move it as close to his hand as possible, respectively.

Explore thumb opposition by asking the subject to touch the tip of each of the rest of the fingers on the same hand with his thumb.

Palpation

Each and every joint of the hand will be carefully palpated, looking for pain.

With the person's fingers slightly flexed, use your thumb and index finger to palpate the metacarpophalangeal joints, which feel like bumps, just at the distal end of the metacarpals. The interphalangeal joints are then palpated.

Muscle strength assessment

Explore the strength of finger extension by placing your hand over the person's closed fist before the hand opens, and the strength of extension by placing your fingers against the palmar aspect of the subject's fingers, opposing the hand from closing.

To explore abductor strength, place your thumb against the subject's index finger and your other fingers against the little finger.

hip joint

anatomical count

The hip joint has a peculiar importance, due to its deep location and its support function.
Covered by thick and powerful muscles, it is difficult to perceive swellings and deformities. Because it is a support joint, it is usually the seat of degenerative processes (osteoarthritis); In addition, the study of gait has semiological value, which was already explained previously.

mind.

The bony elements of the coxofemoral joint are the cotyloid cavity of the hip and the head, and the neck of the femur.

The cotyloid cavity has a spherical shape and is surrounded by the cotyloid eyebrow, which occupies its entire contour, except for the ischiopubic notch, in the lower part; Following this notch is the fossa of the acetabulum. The upper part of the cotyloid brow is the roof of the acetabulum. The head of the femur adapts to said cavity; It is rounded, which is important in the imaging aspect, because it does not vary in any position. The articular surface has only a small depression, the fossa of the round ligament.

The neck of the femur is an irregularly cylindrical bone mass that runs from the truncated part of the head to the vertical portion of the bone, and has a length of around 40 mm. On its external surface and upwards it continues with the greater trochanter, a palpable bony eminence. The lesser trochanter is located in the lower and posterior part of the neck. The axis of the neck and the shaft of the femur form an angle of 130°.

The cartilage covers the entire articular surface of the acetabulum and the entire head, except for the lithofossa.

round gament. The acetabular labrum, a fibrocartilaginous formation, enlarges the articular surface and increases the coverage of the head of the femur, thus containing the tendency to escape from the acetabular cavity.

The synovium lines the internal surface of the joint capsule, part of the external surface of the acetabular labrum, the intra-articular neck and the round ligament. It also covers the ischiopubic orifice.

The muscle formations that act in hip movements are:

- Flexors: iliac psoas, sartorius and rectus femoris.
- Extensors: gluteus maximus, posterior fascicles of the gluteus medius, biceps femoris, semitendinosus and semimembranosus.

Abductors: the three glutes (major, median and minor), piriformis, and tensor fascia latae.

Adductors: the pectineus, the minor, median and major adductors and the internal rectus.

- Internal and external rotators.

On the posterior surface, the relief of the buttocks forms a regular and rounded prominence, the buttock, which is limited on the inside by the intergluteal fold and below, by the gluteal fold; above and outside, the anterosuperior and posterosuperior iliac spines.

The gluteal and intergluteal folds are joined by a curved line of upward and outward convexity. In the inferointernal quadrant of the buttock the tuberosity of the ischium can be palpated. In the most external part, another tuberosity can be palpated, that of the greater trochanter. The sciatic nerve is located at the level of the depression between the ischium and the greater trochanter.

The anterior aspect of the hip corresponds to the anterior aspect of the thigh. Its limits are the groin fold, from the anterior superior iliac spine to the pubic spine, above. In this region there are no bone reliefs, but rather muscular ones; We must point out: the adductor medianus and the sartorius. These two muscles intersect forming an angle open upwards, which is the lower vertex of a so-called Scarpa triangle. The bed of this triangle is formed by two muscles: the iliopsoas and the pectineus, and in the space between them are the femoral vessels. Deep in this triangle is the hip joint.

Clinical examination

Inspection

Because the hip joint is essential for walking movements, explore gait and stance during the general inspection. The hip can be examined with the person standing or lying down. The active mobility maneuvers are the same for both positions; However, the person needs greater strength and balance to perform the maneuvers in the standing position.

Mobility

The following active movements should be considered in the physical examination of the hip:

1. Flexion. Approximation of the thigh to the abdomen; the arc of movement is greater than 120°.
2. Extension. It is the reverse movement; reaches 15°, 3.
Abduction. Separation of the thigh from the axis of the body. At least it reaches 45°.
4. Adduction. It is the reverse movement of the previous one. The thigh should be slightly flexed and reach 30°.
5. Rotation on its axis. We observe the tip of the foot, which runs through a 90° arc.
6. Internal rotation. The arc of internal rotation is about 30°
7. External rotation. Its normal arc is 40°.

To test for hip flexion, ask the person to raise the leg without bending the knee, if tested lying down, supine, or to bring the thigh closer to the abdomen, if standing. Observe the extension, with the subject in a prone position, asking him to raise the leg again, without bending the knee,

To explore abduction, instruct the subject to separate the entire lower limb and adduction from the axis of the body, asking them to move the entire lower limb, crossing the midline, so that the leg passes over the other,

For internal rotation, have him turn his foot inward, also with his leg extended; or in a prone position, with the leg bent and outward, it is rotated inward. To explore external rotation, ask the person to rotate the foot outward while keeping the leg extended; or in a prone position, with the leg bent and inward, it is rotated outward.

Palpation

Palpate the hip joint and surrounding structures. Place the tips of your fingers on the lateral aspects of the iliac crest, with the palm of your hands on the lateral hip. The greater trochanter of the femur and the non-palpable trochanteric bursa lie behind the surface of your palm. Palpate around this process and note any joint swelling or pain. Palpate the surrounding area: the hip, thigh and buttock muscles.

Muscle strength assessment

Assess flexor muscle strength by placing your hand on the anterior surface of the thigh and asking the person to repeat the hip flexion movement.

Explore extensor muscle strength by placing your hand on the back surface of your thigh as you repeat the hip extension movement.

Apply resistance to the lateral aspect of the leg during abduction and to the medial aspect of the leg during adduction to test muscle strength. You can also apply resistance against the lateral and medial processes of the ankle, while abducting and adducting, respectively.

knee joint

anatomical count

The knee is made up of the distal end of the femur, the proximal end of the tibia and the patella. The distal end of the femur is formed by the trochlea, a pulley-shaped surface with a lower concavity and slightly inclined from front to back, and the internal and external condyles, on the sides and back, separated in front by the trochlea and behind, by the intercondylar notch.

The proximal end of the tibia is composed of two glenoid cavities, which correspond to the femoral condyles. Between them is the tibial spine and two tubercles, one internal and the other external. This interglenoid space corresponds to the intercondylar space.

The patella, located in the anterior region of the knee, has a triangular shape with an inferior vertex, where the patellar ligament is inserted; The quadriceps tendon inserts at the base and articulates behind with the femoral trochlea.

The upper end of the fibula, although not part of the knee joint, is located in this region.

The joint capsule is a fibrous sleeve that is inserted into the corresponding bone, outside the articular cartilage.

The synovial membrane extends from the superior edge of the femoral trochlea, runs upward along the anterior surface of the femur, and then reflects downward, lying below the quadriceps, to form the subquadricipital fornix. It is interrupted at the level of the patella and continues below it, lining the posterior part of the anterior adipose bundle of the knee, which separates it from the patellar tendon, and ends at the tibia. On both sides of the kneecap it forms other fornices. The synovium also covers the anterior and lateral faces of the cruciate ligaments, leaving them outside the joint cavity.

On the rest of its surface it covers the joint capsule.

In the knee there are ligaments that keep the joint firm, joining the femur to the tibia:

- Anterior ligament or patellar tendon.

-Lateral ligaments (internal and external).

- Posterior ligament.

- Cruciate ligaments (outside the cavity).

The menisci are two crescent-shaped fibrocartilages, one medial and one lateral, between the articular surfaces of the glenoid cavities and the femoral condyles, whose objective is to facilitate joint adaptation.

They are also interested in this joint, the serous bags. The prepatellar bursae (three, practically overlapping) are located between the patella and the patellar tendon, and the subcutaneous cellular tissue. Anserine bursa, between the muscles that form the "goose foot", the tibial surface and the internal lateral ligament.

In the popliteal fossa there are the bursa for the internal twin, the common bursa for the internal and semimembranous twin (where the Baker cyst develops), and the bursa for the semimembranous twin; In the lateral half are the biceps bag, the external lateral ligament bag and the external gastrocnemius bag, together called the popliteal bag.

Of interest in pathology is the anterior adipose bundle, behind the patellar ligament.

Exploratory anatomy

On the anterior surface of the knee, the relief of the patella stands out. With the leg extended, the joint interline is projected at the level of a horizontal line that passes above the inferior vertex of the patella. Below the kneecap, the patellar tendon is palpated. The subquadricipital fornix projects about 5 cm above the superior border of the patella.

In the most anterior part of the lateral and medial aspects of the knee and above the joint interline, the femoral condyles are palpated. Behind the relief of the internal condyle are the tendons of the pes pes (medial rectus, sartorius and semitendinosus); This tendon set is separated by the anserine bursa.

On the lateral aspect, the head of the fibula is prominent, where the biceps tendon is inserted, which is easily palpated.

In the posterior part, a rhomboidal-shaped gap, the popliteal fossa, is limited above and inside by the semitendinosus muscle; above and outside, the biceps; below and inside, the internal twin; and, below and outside, the external calf. This structure is of great importance, because it houses the popliteal artery and vein, the internal sciatic-popliteal and external sciatic-popliteal nerves; There are also lymph nodes.

Clinical examination

It is most easily explored with the person sitting, with the hips and knees flexed. However, it can be performed with the subject lying down.

Inspection

The knees are inspected from the moment you examine the standing attitude, in the general physical examination.

Inspect the knees from the front and observe their alignment, their usual reliefs and depressions, whether there are deformities, the contour of the quadriceps muscle, and whether or not there is atrophy of this muscle.

Slight knee valgus is physiological; Normally the axes of the thigh and leg form an angle open outwards of 175°. When we observe an inferior angle we can speak of pathological genu valgus and if it is greater than 175°, of pathological genu varus.

The normal contour of the knee is indicated by the depressions on each side of the patella, which will be lost if there is any inflammatory process, with or without synovial effusion.

Mobility

The femorotibial joint is a trochlearthrosis and fundamentally performs flexion-extension movements.

To explore flexion, ask the person to stand and bend the knee, bringing the heel against the glutes. If the person is lying down, to perform this action the hip must also flex. Observe extension, when the person returns the knee to its neutral position.

During flexion, at least 130° is reached, which is limited to the contact of the leg with the thigh, and in extension, the leg is placed in the same axis of the thigh.

To test the muscle strength of the knee during flexion, place your hand against the back of the ankle. During extension, move your hand to the front of the ankle.

Lateral flexion and rotation movements cannot be performed with the leg extended, because of the ligaments that limit these movements. At 90° of flexion, slight lateral flexion and 50° rotation (10° inwards and 40° outwards) are possible. In minimal flexion, both movements are very limited.

Palpation

Palpation is performed in search of temperature changes and bone alterations; The patella and greater tuberosity of the tibia are examined, and subsequently the synovial ligaments.

Feel the temperature of the knee with the back of your hand, comparing it with that of the knee on the other side and with the thigh and leg on the same side. At the level of the kneecap, the skin is less warm than on the thigh and leg.

Focus palpation on the suprapatellar bursa, a sac-like structure that separates the patella from surrounding structures. The accumulation of fluid can be easily detected in it, due to trauma or illness.

To help make the fluid more accessible to palpation, move the bursa downward, placing one of your hands on the quadriceps above the knee and applying slight downward pressure. Use your other hand to palpate each side of the patella and over the tibiofemoral joint space.

Palpate each side of the patella with your fingertips and stabilize the opposite side with the thumb of your examining hand.

Next, move the bursa upward by placing your hand on the bottom of the knee and applying light upward and inward pressure. Palpate the area from the quadriceps to the patella, with the fingertips of the other hand. Look for any swelling, edema, sinking, or thickening.

If you suspect an effusion, perform the special exploration maneuvers, which, together with the maneuvers to explore the ligaments, will be described in Section II.

Also take the opportunity to palpate the popliteal pulse, located behind the knee, in the popliteal fossa, slightly lateral to the midline. Afterwards, you can also explore the patellar or patellar reflex (fourth lumbar root).

Ankle examination

anatomical count

This joint is formed by the distal ends of the tibia and fibula and the talus; The lower end of the fibula is located outside that of the tibia, it constitutes the external malleolus.

The lower end of the tibia articulates with the fibula on its external surface; On its lower surface it adapts to the talar pulley of the fibula; The inner surface constitutes the inner malleolus.

The talus is below the bones of the leg and above the calcaneus, to which it transmits the weight of the body. It consists of three parts: the posterior or body, which constitutes 3/4 parts of the bone, the median or neck and the anterior or head. The upper surface of the talus is shaped like a pulley and articulates with the tibia. The head of the talus articulates with the scaphoid, the lower surface of the bone articulates with the calcaneus.

Like almost all joints, it has a capsulosynovial formation, but in its anterior and posterior part it is quite lax, to allow joint movement.

It presents the internal lateral ligament or deltoid and the external lateral ligament.

The tendons of the leg muscles that act as flexors and extensors of the foot slide in the ankle region.

Exploratory anatomy

The anterior region has a convex shape, in a transverse sense, and concave in a longitudinal sense. If the foot is in active dorsiflexion, we palpate the tendons of this region, from outside to inside, the common extensor of

the fingers, the extensor of the big toe and the tibialis anterior, which is the thickest.

The internal malleolus is more anterior and superior than the external malleolus. A horizontal line that passes one centimeter above the vertex of the medial malleolus corresponds to the tibiotarsal joint interline. Behind the malleoli, two regions are distinguished: posterointernal, between the internal malleolus and the relief of the Achilles tendon, and posteroexternal, between this tendon and the external malleolus; They are the retromalleolar canals. In the internal canal the beating of the posterior tibial artery can be heard.

Clinical examination

The ankle is examined with the person sitting or, preferably, supine, for the anterior region, and prone, for the posterior; In this last position, it is advisable for the foot to hang over the edge of the examination table or bed.

Inspection

Compare the contour of both ankles to rule out the existence of inflammation or deformities.

Mobility

This joint is a trochlearthrosis, so the almost exclusive movements are flexion and extension.

Flexion brings the dorsum of the foot closer to the leg and extension moves it further away. Maximum dorsiflexion can reach 75° and plantar flexion can reach 145°.

To evaluate ankle dorsiflexion, ask the person to bend the ankle, directing the toes toward the knees. Observe the range of motion (normal: 20°).

Explore plantar flexion by asking the subject to point the fingers (normal: 45°).

To explore ankle inversion, ask the subject to turn the sole of the foot inward at the ankle joint (normal: 45°).

Explore ankle eversion, while the person turns the sole outward at the ankle joint (normal: 30°).

Palpation

To palpate, stabilize the ankle by placing one of your hands behind the heel. Palpate with the fingers of the other hand.

Also take the opportunity to palpate the posterior tibial pulse, located slightly below or behind the tibial malleolus. Also explore the Achilles reflex.

Muscle strength assessment

Assess the resistance to dorsiflexion, with your hand on the dorsal surface of the foot.

Assess muscle strength in plantar flexion, applying resistance with the hand on the sole of the foot and to In the case of ankle eversion, apply your hand against the fifth metatarsal bones.

Foot examination

The physical examination of the foot is of great importance, since it is the support organ, and its importance is essential for walking. The foot is pointed with the toe outward (external rotation), about 15° away from the midline.

anatomical count

The foot is made up of several small bones tightly joined by powerful ligamentous structures, to fulfill the objective of supporting the weight of the body.

The talus has already been considered when studying the tibiofibular-talar joint. Below the talus, the calcaneus supports much of the body's weight. The lower surface of the calcaneus rests directly on the ground and on it we observe three eminences: two posterior ones, the internal and external tuberosities, and one anterior.
Some muscles of the sole and ligaments are inserted into them. In front, the calcaneus ends in a kind of extension that articulates with the cuboid.

The talus and calcaneus constitute the posterior part of the tarsus, while the cuboid, scaphoid and the three wedges constitute the anterior part. The cuboid is the most externally located bone. It articulates with the calcaneus behind, in front with the fourth and fifth metatarsals, and inside with the third cuneiform.

The scaphoid articulates posteriorly with the head of the talus and anteriorly, with the three wedges.

The first, second and third wedges articulate with the scaphoid posteriorly and with the first four metatarsals in front.

The metatarsals, in number of five, articulate with the tarsal bones at their posterior end and at their anterior end, with the phalanges of the corresponding fingers; The posterior ends also articulate with each other. They are called first, second, through fifth, metatarsal, from inside to outside. The heads of the five metatarsals form an arch of inferior concavity (transverse arch).

The skeleton of the fingers is made up of the phalanges: first, second and third, from back to front (from the proximal to the distal portion). The first finger, or big toe, only has two phalanges, first and third.
The rest of the toes do not have specific names like those on the hand. They are named by the place they occupy, from the inside out (second, third, fourth and fifth fingers).

The foot is formed by three arches: internal longitudinal arch (calcaneus, talus and scaphoid, first cuneiform and first metatarsal), the external longitudinal arch (calca-

neo, cuboid and fifth metatarsal) and the transverse arch (heads of the five metatarsals).

The joints of the foot are as follows:

- Subtalar joint.
- Chopart's midtarsal joint.
- Tarsometatarsal and tarsal joints.
- Metatarsophalangeal joints.
- Interphalangeal joints.

The musculotendinous formations are made up of the tendons of the leg muscles and the muscles of the foot. These formations contribute to maintaining the statics of the foot, adapting to the situation at all times.

The motor action of the foot muscles is complex;
We are going to present it schematically, for your better understanding.

The triceps is a plantar flexor, adductor and supinator. The flexor action is carried out on the internal edge; Adduction and supination are a consequence of the arrangement of the articular facets of the subtalar joint. The peroneus longus is a plantar flexor, abductor and pronator; flexes acting on the left edge. The action of these two muscles determines plantar flexion.

The tibialis anterior is a dorsiflexor of the foot, adductor and supinator. The common extensor of the toes is the dorsiflexor of the foot and acts on the toes.

Exploratory anatomy

Three sides of the foot are studied: internal, dorsal and plantar.

On the sole of the foot there are dense aponeurosis and fibrous formations. It has a domed shape and is supported by its external, anterior and heel edges. Normally, the inner edge does not contact the ground. The toes are shorter than the fingers. The first finger is the largest.

Foot statics

The foot rests on: plantar tuberosities of the calcaneus, the head of the first metatarsal and the head of the fifth metatarsal. These support points are joined together by the three bony arches that support the plantar dome. The support points are separated from the ground by soft parts. The external longitudinal arch and the anterior arch are also separated from the floor by soft parts. At the level of the internal arch the skin does not contact the ground.

The plantar dome is supported by:

- Special arrangement of the bones.
- Powerful ligaments.
- Action of musculotendinous formations.

Clinical examination

Inspection

Inspect the feet and observe the integrity of the skin, conditions of the nails and whether there are alterations that deviate from their normal morphology, their position with respect to the midline, the contact relationship of each of their parts with the horizontal plane, the degree of abduction or adduction that they present, as well as active motility.

With the person standing on a hard surface, observe

mos:

1. If there is parallelism of the feet: normally, the tips of the feet are almost parallel, slightly diverging outwards at an angle of 15°.
2. Whether the height of the internal longitudinal arch is normal or not (it is a visual practice problem).
3. If the heads of the metatarsals are arranged normally, in the form of a convex arc, dorsally.
4. If the Achilles heel and the posterior edge of the heel, observing the subject from behind, are located on the same line.
5. We can also observe the respective position of the malleoli.

Mobility

Foot mobility is very poor; It is not measured in degrees; It is only important to check whether they are present or not.

The foot can perform adduction and abduction movements, as well as pronosupination.

In adduction, the forefoot is directed inward; abduction is the opposite movement.

In supination, the plant faces inward; In pronation, it performs the opposite movement.

These movements are carried out together. Adduction is combined with supination (inversion movement) and abduction with pronation (evertion movement).

Stabilize your foot by cupping one of your hands around the heel. Ask him to turn his foot in (adduction) and out (abduction).

Assess finger flexion and extension by asking the person to bend and straighten them, respectively.

The metatarsophalangeal joints of the first toe have flexion and extension movements that reach 90°, 45° dorsally and 45° plantarly.

To explore abduction of the fingers, ask them to fan them out, and for adduction, observe the return of the fingers to their neutral position.

Palpation

It is done with the subject sitting. The first thing we will do is observe whether palpation is painful or not, and then we will practice passive movements.

Palpate, grasping between the index finger and thumb, the bones and metatarsal joints. Take the opportunity to palpate the dorsal pedis pulse, which can be felt just lateral to the extensor tendon of the first finger.

APPROACH TO THE WRITTEN RECORD OF THE SOMA EXAM

- Structural symmetry and alignment.
-Ease and range of movement.
- Muscle strength and muscle tone.
- Muscle mass.
- Appearance of the skin over the joints.
- Pain, crepitation and deformities.

REGISTRATION OF THE NORMAL OSTEOMIOARTICULAR EXAMINATION

Fluid and coordinated gait; symmetrical limbs and correct posture. Range of movements required for general movements during the examination, intact; no specific maneuvers are made.

Preserved muscle strength; well-developed muscle masses. No visible or palpable osteomyoarticular deformities.

8

CLINICAL ANATOMY AND PHYSIOLOGY OF THE CHEST AND RESPIRATORY SYSTEM

CLINICAL ANATOMY

The thorax has a conical shape with a depressed lower base in an anteroposterior

The rib cage is formed:

Past	Breastbone Sternocostal joint Anterior extremity of the ribs
Sideways	costal arches
Behind	Dorsal vertebrae Costovertebral joint Posterior end of the ribs

There are 12 ribs in each hemithorax. Occasionally there are supernumerary ribs dependent on the seventh cervical vertebra, which, causing compressive symptoms, can lead to false diagnoses with:

1. Tumors in the supraclavicular region.
2. Circulatory disorders due to compression of the subclavian artery.
3. Nervous disorders due to compression of the brachial plexus.

The intercostal spaces are wider in the anterior part of the thorax; This detail is important when we study pleural effusions, pneumothorax, exploratory punctures, etc. The internal mammary artery is located half an inch from the sternum, at the level of the second and third costal arches. At this point it is located to tie it in case of injury or rupture. The intercostal vessels and nerves are housed in the costal sulcus (lower edge of the rib); At the level of the posterior axillary line they occupy a middle position in space, so there is a risk of injuring them during punctures.

Due to the obliquity of the ribs, the anterior costal extremity is below the posterior costal extremity. In emphysema, for example, the ribs adopt a horizontal position.

The thoracic vertebrae as a whole form a curvature of posterior convexity, sometimes slightly deviated to the right by the

greater use of the upper extremity on that side or possibly due to the pressure exerted by the thoracic aorta. On each side of them are the costovertebral spaces whose bottom is made up of the transverse processes and the posterior costal extremity. The spinous process of the seventh cervical vertebra is very prominent and serves as a reference point to locate the rest of the dorsal column. The spinous processes of the first and second dorsal vertebrae are also prominent and relate directly to the body of their corresponding vertebra. The rest of the spinous processes are directed downwards and each one is related to the body of the immediately lower vertebra, a detail to be taken into account to locate the affected areas by palpation. Starting from the twelfth dorsal vertebra, the spinous processes become horizontal again and relate directly to their corresponding vertebral body.

The thoracic bone cage is covered on the outside, by:

- bone formations
  - Clavicles
  - Shoulder blades
- muscle formations
  - Former
    - Pectorals
    - Deltoid
  - Later
    - Spinals
    - Supraspinatus
    - Infraspinatus
    - Serratus major
    - Dorsal latissimus
    - Trapezius
    - and

Inside, the musculoskeletal portion is surrounded by the parietal pleura, which at the level of the pulmonary hilum folds back to cover the lungs, and is then called the visceral pleura. Between both pleurae there is a virtual space, the pleural cavity. Normally both pleurae slide over each other, but in adults it is common to find bands of adhesions, residues of old pathological processes.

The area between the right and left lungs is called the mediastinum. Both pleural cavities are separated by the mediastinum, which is covered by the visceral pleura, which is called the mediastinal pleura, which in the anterior portion is reflected forming a potential space that constitutes the costomediastinal sinus. The same happens at the level of the diaphragm, also covered by the pleura, diaphragmatic pleura, and when it comes into contact with the parietal

They form the costophrenic sinuses. The pleural space is occupied by a small amount of serous fluid that acts as a lubricant and allows the sliding of both pleural sheets. Any injury that affects the pleura interferes with this sliding and causes true friction during the respiratory act (figs. 8.1, 8.2 and 8.3).

The visceral pleura receives innervation from the vagus and the sympathetic through the lung fillets. There is no painful sensitivity at this level. The parietal pleura, in addition to these nerve bundles, also receives them from the phrenic and the intercostals, some of which have fibers sensitive to painful stimuli. For this reason, conditions that attack the parietal pleura are usually extremely painful.

The right diaphragmatic dome is located higher than the left and rejected upward by the right lobe of the liver.

The lungs are elastic, spongy structures that are fixed to the mediastinum by the pulmonary hilum, through which the arterial and venous vessels and the large bronchi enter and exit. Despite the diaphragmatic elevation, the right lung is longer and wider than the left.

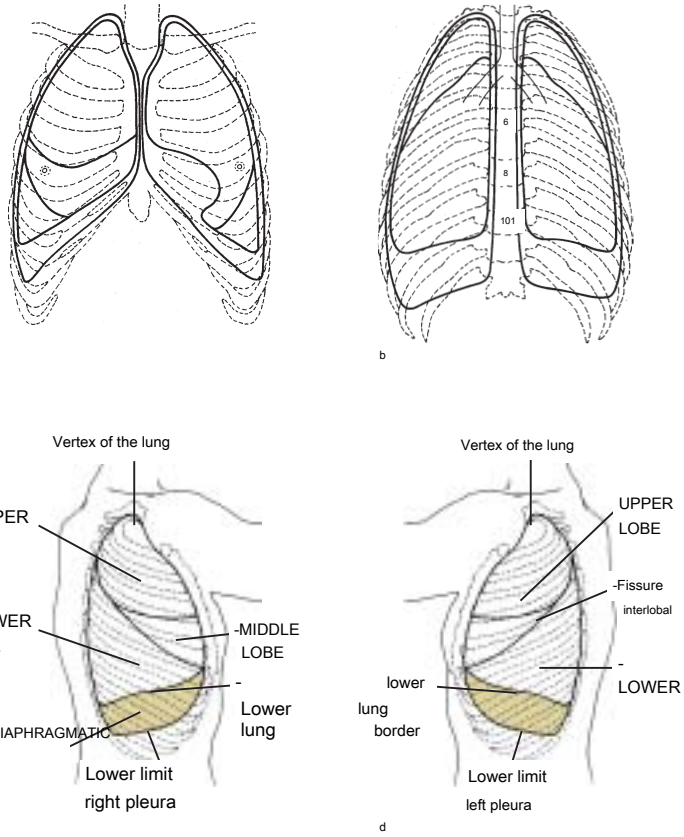
The left lung is divided into two lobes by a deep fissure that penetrates to the hilum and runs obliquely from the posterior wall below the pulmonary apex, downward and forward, until reaching the anterior edge. It can be said that the upper lobe includes the vertex and much of the anterior portion of the lung, while the lower lobe includes the base and the posterior portion of the lung.

The right lung is also divided by the fissure into two large lobes, but, in addition, an additional fissure appears that extends horizontally from the middle of the large fissure, to the anterior edge, which constitutes the middle lobe, which together with the lower one forms the lung base.

Laterally, the right pulmonary apex is in direct contact with the trachea, while the subclavian is interposed on the left. On the right side, the subclavian is in front of the vertex, while on the left its position is more medial. On the right side, the vena cava and the brachiocephalic trunk are located in front of the middle portion of the vertex. All these different relationships between the pulmonary vertices and the structures that surround them confer special characteristics to the exploratory signs that can be obtained at this level, which is why they are not comparable to each other.

The trachea extends from the lower orifice of the larynx to its bifurcation at the level of the second chondrosternal joint. Throughout its entire path it occupies the outer midline.

Fig. 8.1 Relationship of the pleura, lungs and fissure with the chest wall: a, anterior chest wall; b, posterior chest wall; c, right chest wall; d, left chest wall.



except at its lower extremity, where it is located slightly to the right; hence the right bronchus is considered the continuation of the trachea. The right main bronchus is shorter, wider and more vertically aligned than the left one, which is why most of the foreign bodies aspirated through the trachea are located in this bronchus. Approximately half of the trachea is located in the neck and the other half in the thorax, where it establishes relationships:

To the right	Superior vena cava Azygos vein Vagus nerve
To the left	arch of the aorta Recurrent or inferior laryngeal nerve left subclavian artery

Fig. 8.2 Visceral projections in dorsal position.

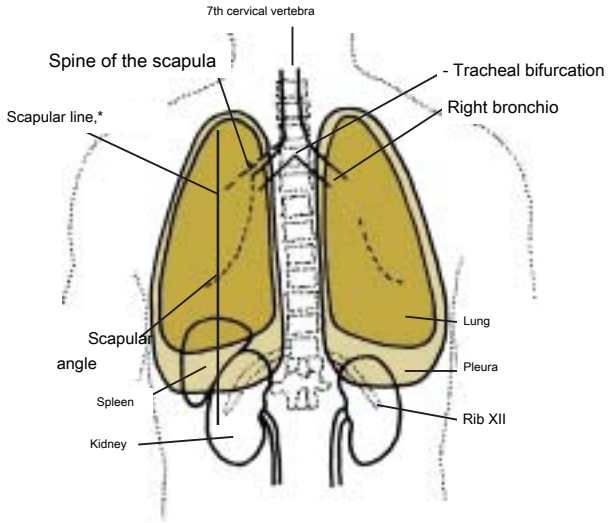
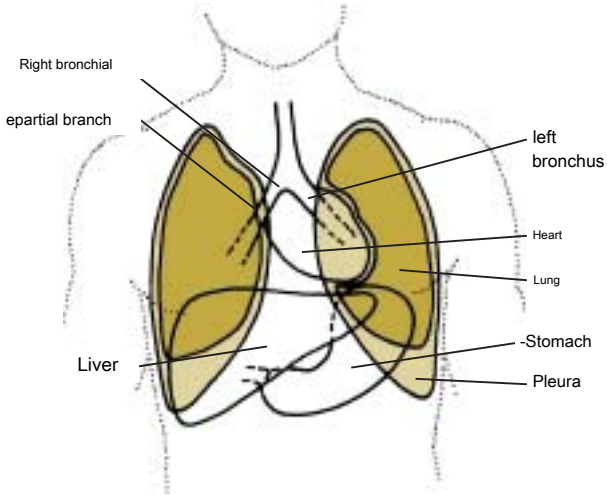


Fig. 8.3 Visceral projections in frontal position.



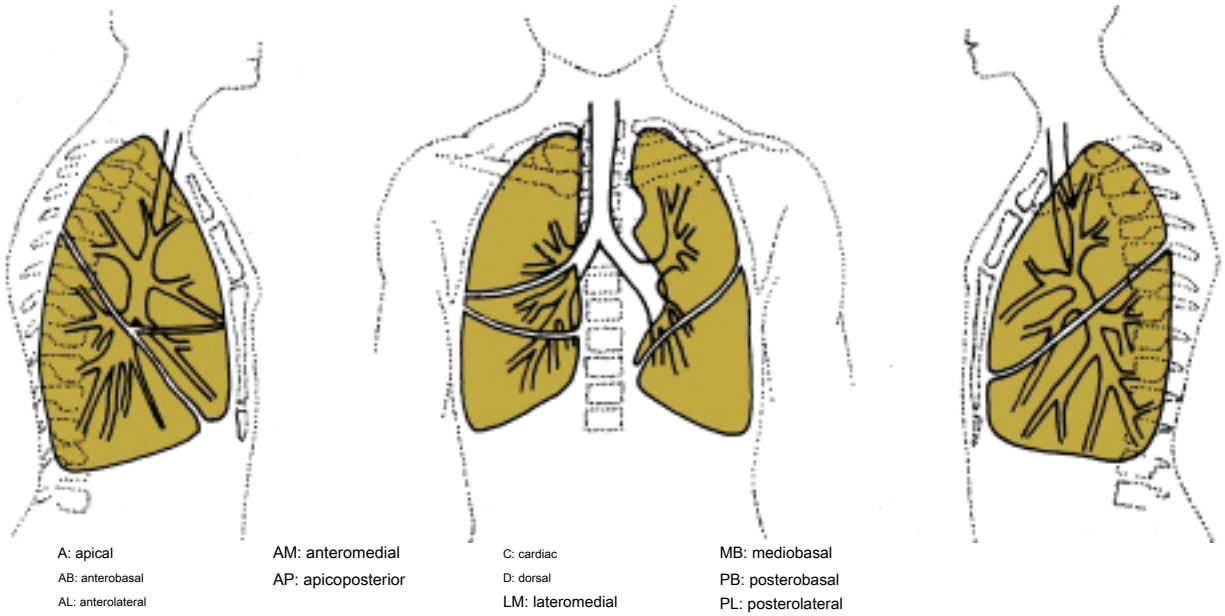


Fig. 8.4 Bronchial distribution (Foster-Carter).

In front, at the level of the bifurcation, is the birth of the arch of the aorta.

The bronchi divide into secondary branches, which enter the lung at the hilum. The right bronchus gives rise to several branches: one for the upper lobe, an eparterial branch because it originates above the pulmonary artery; The rest of the branches (the one from the middle lobe and the one from the lower lobe) arise below the artery and are called hyparterial. The left bronchus, in turn, only gives rise to two hyperarterial branches.

The bronchial tree is thus divided into a series of branches corresponding to segments or wedges of lung tissue named according to their projection:

Right bronchus-trunk	Upper lobe bronchus Middle lobe bronchus Lower lobe bronchus
Left bronchus-trunk	Upper lobe bronchus Lower lobe bronchus

In turn, each bronchus-trunk gives rise to a series of branches that constitute the segmental bronchi, which serve as identification units for the exact location of respiratory processes. Currently, the internationally accepted classification is the Foster-Carter classification (fig. 8.4), which with some language modifications is the one we use in our environment.

In this way, the bronchial tree is divided into three

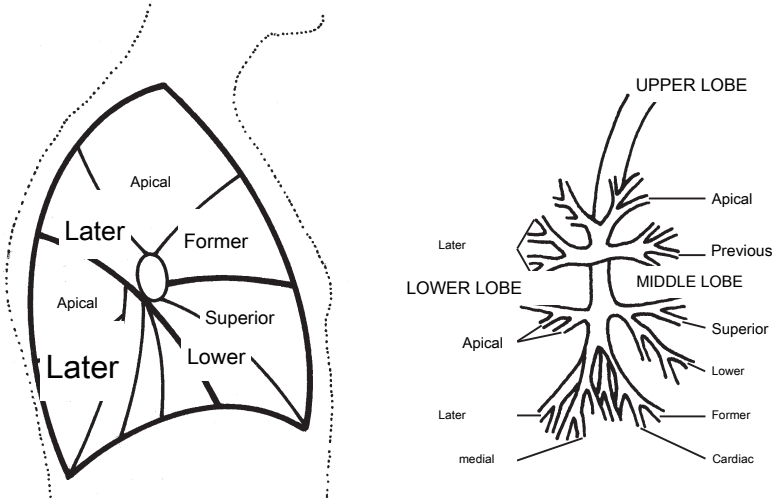
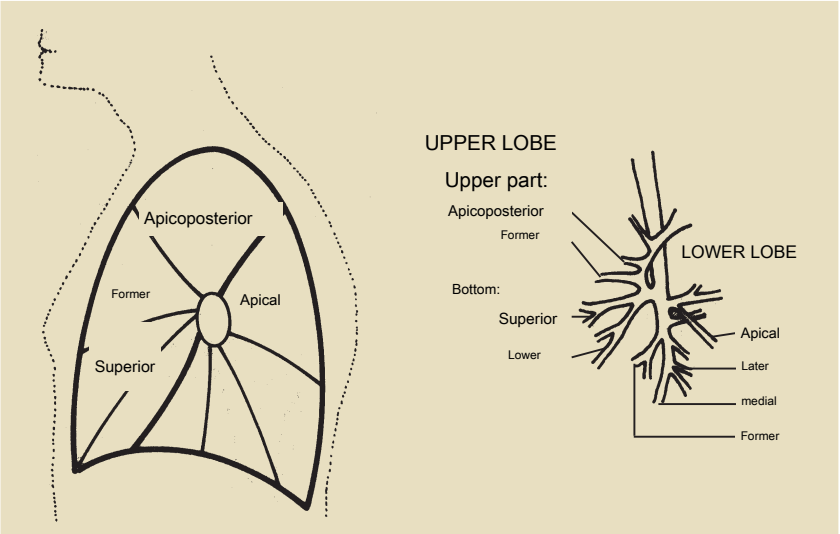


Fig. 8.5 Right bronchial distribution.

Fig. 8.6 Left bronchial distribution.



lobes for the right lung: upper, middle and lower; and two for the left lung: upper and lower (figs. 8.5 and 8.6).

RIGHT LUNG Lobes
Segments

Apical
Superior Posterior
Former

Superior
Half
Lower

Posterior
Apical
Lower medial
Cardiac
Anterior

LEFT LUNG Lobes
Segments |Apicoposterior |

Part superior
Anterior

Superior

Part JUpper and
lower |Inferior

Lower

Apical
Posterior
Medial
|Previous

The bronchi continue gradually narrowing to bronchioles and go into the alveoli, in the lungs.

Each bronchial branch continues to subdivide until it reaches the bronchi of 1 mm in diameter, at which level the cartilage is lost, constituting the respiratory bronchioles, from which the alveolar ducts emerge.
Each alveolar duct gives rise to a series of saccules called atria, which are the points of origin of the alveoli or respiratory cells. The respiratory bronchiole, the atria and the alveoli, together with the blood vessels, lymphatics and nerves that surround them, constitute the pulmonary lobules; that is, the indivisible units of the lung. Within these lobules Capillaries come into close relationship with the alveolar walls, allowing the exchange of respiratory gases (figs. 8.7 and 8.8).

The blood supply to the lung is derived from the bronchial vessels and, to a certain extent, from the minor or pulmonary circulation through which hematosis occurs, by diverting venous blood from the right heart to the lung through the pulmonary artery and collecting the oxygenated blood through the pulmonary veins, to empty it into the left heart. It must be taken into account that the bronchial arteries end their journey at the level of the respiratory bronchi and this blood is returned through the bronchial veins without becoming oxygenated, which is why arterial saturation is always incomplete.

The innervation of the lung is derived from the vagus and sympathetic nerves. There are no painful sensory fibers, for this reason it is common to observe advanced pulmonary processes without major pain manifestations.

The mediastinum separates both lungs. It is covered laterally by the parietal pleurae: in front it has the sternum and behind the vertebral column.

It serves as a transit route for large vessels, nerves and organs of passage such as the esophagus and trachea. It is divided into superior mediastinum and inferior mediastinum and the latter into anterior, middle and posterior mediastinum.

The superior mediastinum extends from the upper thoracic opening to the tracheal angle at the level of the fourth thoracic vertebra. It contains the lower portion of the trachea, part of the esophagus, the remains of the thymus, the arch of the aorta and the three large arterial trunks that emanate from it; the upper part of the vena cava, the brachiocephalic trunk and the origins of the subclavian, the thoracic duct, the vagus, the left laryngeal recurrent and the phrenic nerves.

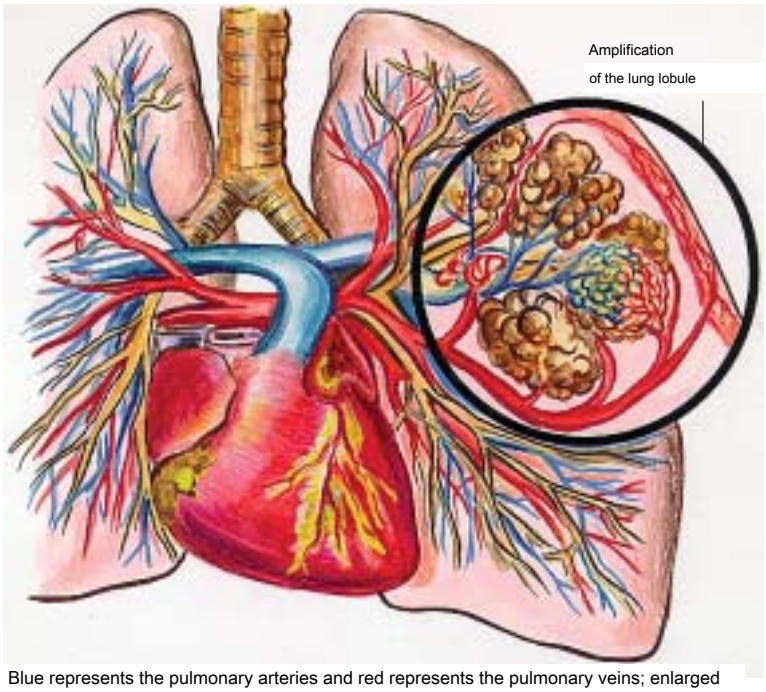


Fig. 8.7 Functional unit of the lung.

Blue represents the pulmonary arteries and red represents the pulmonary veins; enlarged in red, the bronchial arteries that supply the interstitial stroma and pleura.

The anterior mediastinum extends from the tracheal angle to the diaphragm; Its anterior limit is the sternum and its posterior limit is the pericardium; laterally, the parietal pleurae. It only contains areolar tissue and some lymph nodes.

The middle mediastinum has the same extension as the anterior one, but its anterior and posterior limits are formed by the pericardium. It contains the heart, the ascending aorta, the large bronchi, the pulmonary arteries and veins, the inferior portion of the superior vena cava, and the mouth of the azygos vein. The phrenic nerves slide between the sheets of the pericardium and the parietal pleura. Numerous nodes are found surrounding the bronchial structures.

The posterior mediastinum extends from the fourth thoracic vertebra to the diaphragm, contains part of the esophagus, the descending aorta, the thoracic duct and the azygos veins, as well as numerous nodes.

Such important structures housed in such a small space mean that any abnormal growth (aneurysm, tumors, abscesses, etc.) causes very obvious and annoying symptoms.

The lymphatics play a great role in the spread and progress of a large number of diseases, as well as in healing or healing processes, examples: tuberculosis, bacterial pneumonia, etc. Its characteristics are similar to those of blood vessels and inside you can see valves that direct the flow of lymph in certain directions. The lung has an abundant lymphatic irrigation that is distributed between a superficial and a deep network; the first, at the level of the visceral pleura and the second accompanies filling the bronchi, arteries and veins; They communicate with each other, only at the level of the pleura and the pulmonary hilum.

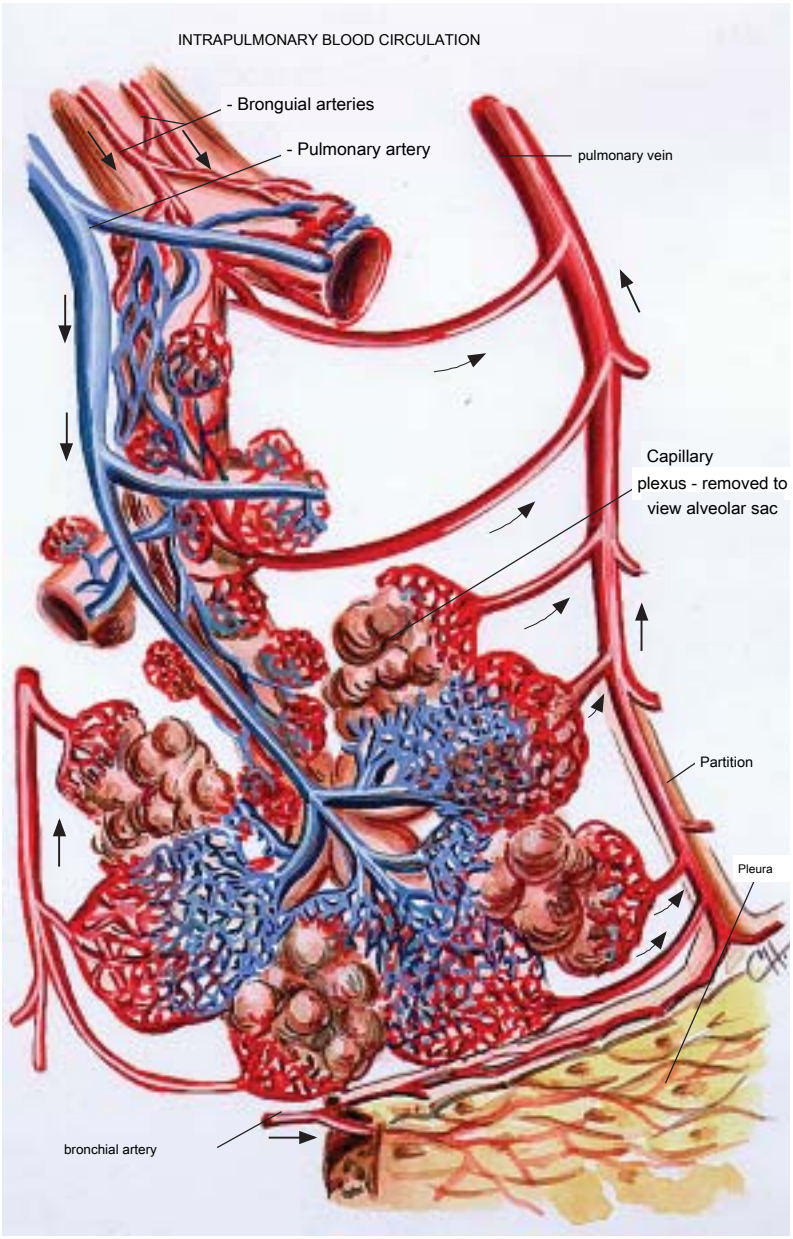
The direction of lymphatic flow is of the greatest importance; The valves allow circulation in only one direction. Apparently the circulation of the arterial and bronchial networks is towards the interior of the lung to the points of emergence of the pulmonary veins, from which they accompany them to the pulmonary hilum. There are no lymphatics at the level of the atria and alveoli, but rather these begin to branch at the level of the pulmonary lobule in the respiratory tract. At the level of the connective tissue existing under the pleura and in the interstitial spaces, they are the points at which the deep network anastomoses with the superficial network, but in such a way that the valves existing there do not allow the lymph from the pleural spaces to penetrate towards the lung and the lymph from the latter can pass to the pleural lymphatics.

The superficial network is very extensive and extends below the visceral pleura; These vessels come together to form thick trunks that, together with those coming from the deep network, flow to the hilar ganglia.

The lung lymphatic tissue is arranged in the form of: lymph nodes, lymph follicles, and small masses of lymphoid tissue. These structures are scattered, but in general terms they tend to converge in certain structures, arranged in the following way:

Locations	Peribronchial Perivenous Periarterial Pleural

Fig. 8.8 Gas exchange.



In general terms, the nodes are found at the bronchial bifurcation points. The pleura never contains lymph nodes.

Small masses of lymphoid tissue, like lymph nodes, act as true filters of lymphatic circulation by accumulating phagocytes loaded with material that cannot be assimilated by the body.

CLINICAL PHYSIOLOGY

The components of the respiratory system begin in the nose and mouth. Air is inhaled through the nose where it is warmed and filtered, before entering the trachea and bronchi and passing to the lungs. The trachea is lined with mucus-producing cells, which trap foreign material, and with cilia (fine hair-like projections) that sweep the mucus upward through the airways. Mucus also moves upward with the cough reflex. Ciliary movements are especially more intense at the tracheal bifurcation or carina, where the trachea branches into the right and left main bronchi.

Respiration is the gas exchange between the organism and the environment that surrounds it. It consists of absorbing O, and eliminating CO_2.

As the blood passes through the lungs, it absorbs O and carries it to the tissues, where the carbon is oxidized to form CO, and the hydrogen is transformed into water. The blood transports CO to the lungs to eliminate it through the exhaled air. This constitutes external or pulmonary respiration.

Internal respiration is a phenomenon that occurs inside the tissues, in which the iron in hemoglobin acts as a catalyst, dissociating the O and making it usable.

The normal subject needs 140 mL of O, per minute, per square meter of body surface. The quantity and quality of hemoglobin (Hb) regulate the contribution of O,.
But the oxygenation capacity of Hb also depends on the physicochemical composition of the blood and the amount of CO it contains.

The normal functioning of the respiratory system relies on the normality of the muscular and neurological systems.

Breathing consists of two times: inspiration and expiration. Both occur as a result of pressure changes within the lungs. The thrusts, into the lungs and out of the chest wall, create a negative pressure that prevents the collapse of the lungs. When the lungs are at rest, the pulmonary pressure is equal to atmospheric pressure. During inspiration, the diaphragm contracts and moves downward.

The external intercostals then push the ribs upward and pulmonary pressure becomes negative, allowing air to enter. When the inspiratory muscles relax, pulmonary pressure becomes positive and air is expelled.

Inspiration is an active process and is due to the contraction of:

The inspiring muscles	Scalene external intercostals
	Serratus
	Diaphragm

The diaphragm is the main muscle used in breathing and is controlled by the phrenic nerves from the third to the fifth cervical vertebrae. Accessory muscles, such as the trapezius, scalenes, and sternocleidomastoids, are used during extra inspiratory efforts; The abdominal and internal intercostal muscles are used in extra expiratory efforts.

Expiration is passive and produced:

Directly	Elastic retraction of the lung	
	Expiratory muscles	Internal intercostals
		Dorsal quadratus
		Triangular of the sternum
		Inferior portion of the serratus major
Indirectly	JMuscles	greater oblique
	abs	minor oblique
		Transverse Anterior
		Rectus

During breathing, the three diameters of the thorax change: the vertical, the anteroposterior and the transverse.

It must be remembered that there is a visceral and a parietal pleura (the latter can be mediastinal costal and diaphragmatic) and that the pleural cavity has a negative pressure of approximately 5-6 mm Hg. Intrapleural negative pressure favors pulmonary circulation and venous return during the inspiratory phase.

Expiration is a passive phenomenon that occurs when the contraction of the diaphragm ends. At this moment, no inspiratory force acts and the expulsion of the air stored in the alveoli is possible thanks to the elastic fibers that surround them like a mesh and that, upon returning to their initial position, compress the alveolar sacs, promoting their emptying.

Figure 8.9 illustrates the arrangement of elastic fibers in the lung lobules and figure 8.10 the state of the alveoli and the lung during inspiration and expiration.

To regulate breathing, there is a bulbar respiratory center that works automatically due to double excitation: nervous and chemical, all regulated by the higher cortical centers. Nervous excitation depends on the Hering-Breuer pneumovagal reflex, through which alveolar distension causes expiration and alveolar collapse causes inspiration.

The afferent or centripetal pathway is made up of the nerves: pneumogastric or vagus, glossopharyngeal, trigeminal, superior laryngeal and cutaneous and visceral sensory organs. The vagus is an inhibitor of inspiration and an excitator of expiration and is also a bronchoconstrictor; the sympathetic is a bronchodilator. The efferent or centrifugal pathway is made up of the nerves: phrenic, respiratory spinal and lower laryngeal.

Chemical excitation is verified by variations in the tension of O and CO in the blood. A high CO content increases blood pH and excites the center, causing faster and deeper breathing.

A low CO content decreases blood pH and depresses the center, therefore breathing becomes less frequent and shallower.

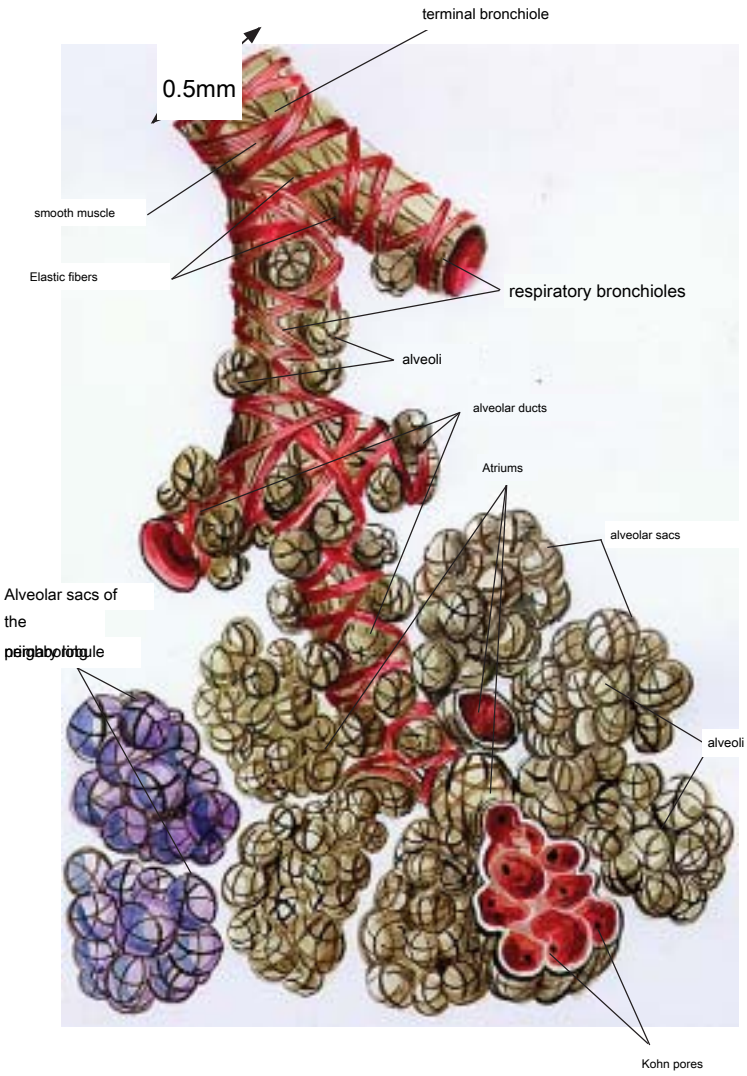
The aorta and the carotid sinus are also starting points for reflexes that act on the respiratory center through mechanical and chemical stimuli.

In summary, we can say that the activity of the respiratory center depends

1. Blood pH.
2. Amount of O, which you receive.

Fig. 8.9 Elastic fibers of the lung.

STRUCTURE OF THE PULMONARY LOBULES



3. Amount of C0, which you receive.

4. Excitability of the center.

5. Metabolism of the center.

6. Hering-Breuer reflex.

PULMONARY VENTILATION (FIG. 8.11)

The compliance of the lungs and chest also affects breathing and involves the ability of the lungs and chest to expand and return to their natural elastic recoil.

Compliance is considered high or low depending on the pressure needed to expand the lungs. For example, if the lungs expand easily, compliance is high, while if more pressure is needed to expand the lung, it is low, in which case the lung feels stiff.

Due to the surface tension of the fluid that lines the alveolus, these small air sacs tend to shrink.

Surfactant, a phospholipid substance, is secreted by the alveoli to decrease surface tension. Decreased surface tension prevents alveolar collapse and reduces breathing effort. Respiratory muscle strength and compliance affect lung volumes, which vary with body size, age, and sex.

Total lung capacity, or the amount of gas in the lungs after a forced inspiration, contains the tidal and residual volumes and the inspiratory and expiratory reserve volumes. The tidal volume is known internationally as Tidal volume, which means tide in English, due to the similarity of the air normally inspired and exhaled with the tide, which comes and goes.

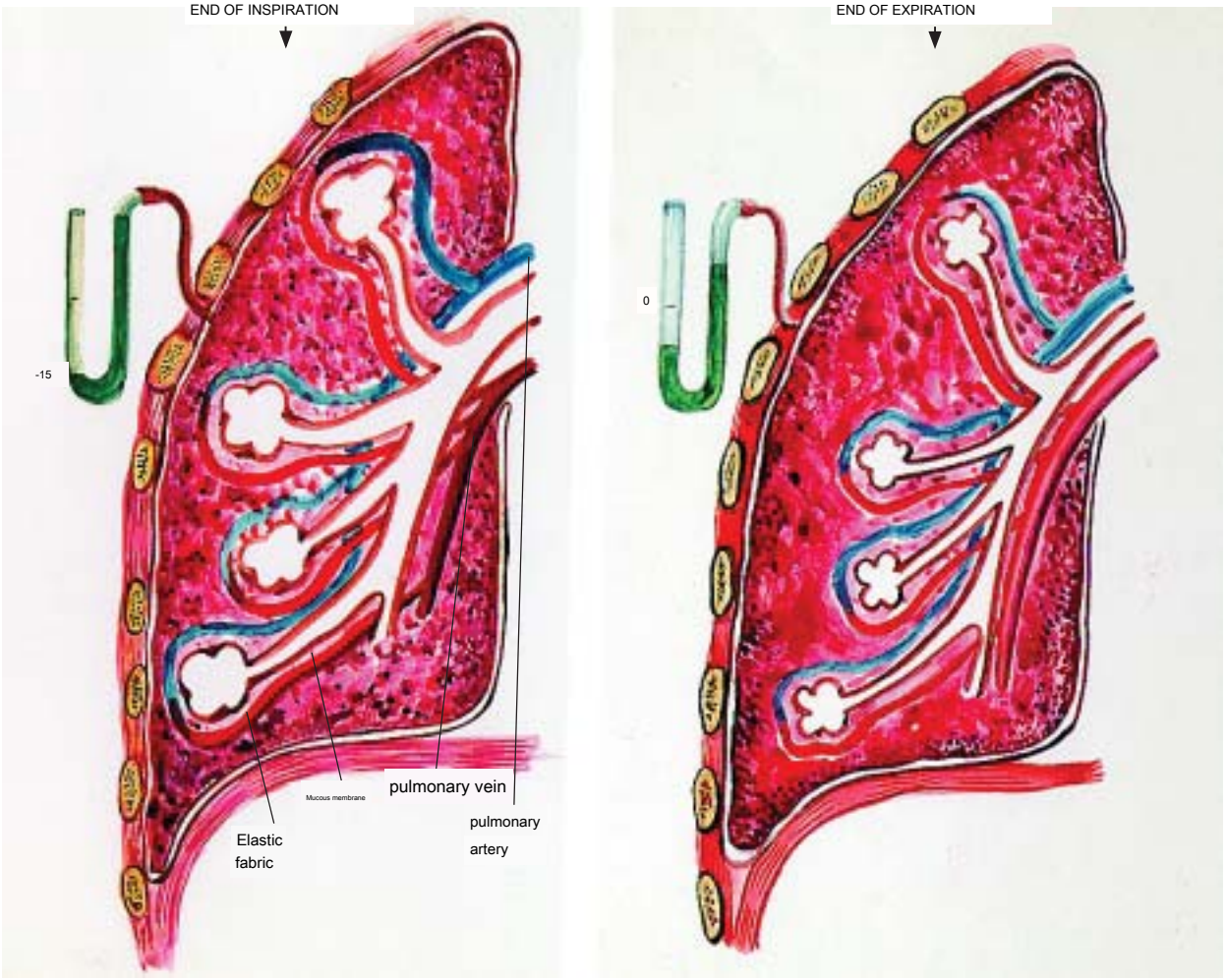


Fig. 8.10 State of the alveoli with inspiration and expiration.

Tidal or "Tidal" volume. Amount of air that enters or leaves the lung with each normal breath; equals 500 mL.

Complementary air or inspiratory reserve volume.
Amount of air that can enter the lung through forced inspiration once normal inspiration has been completed; fluctuates between 1500-2 000 mL.

Reserve air or expiratory reserve volume. Amount of air from a forced expiration after completing normal expiration; equals 1500 mL.

Residual air or residual volume. Amount of air remaining in the lungs after maximum expiration; It is equivalent to 1,000 mL.

Dead space air. The one that remains in the trachea, bronchi, etc.; Its value is equal to 140 mL.

Total capacity. Total air volume; It is equivalent to 4,500 mL.

Vital capacity. Current air + supplementary air + + reserve air = 3 500 mL; It is proportional to the body surface.

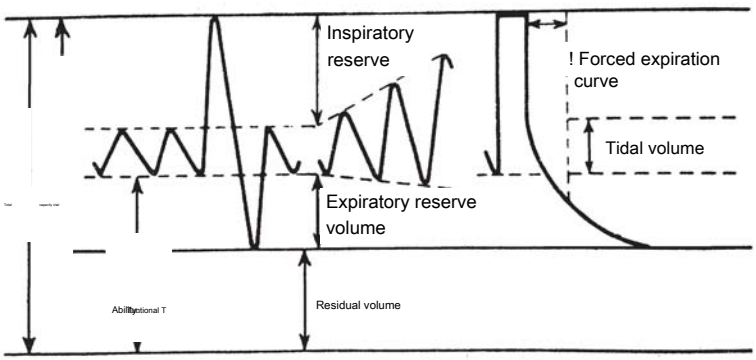


Fig. 8.11 Lung volumes and flows (Simonin, Sadoul and Oriol).

Functional residual air. Residual air + reserve air = =2 500 mL.

gas exchange

Normally there are 15 g of hemoglobin (Hb) in 100 mL of blood. Each gram of hemoglobin fixes 1.34 mL of O,. Therefore, 15 g of hemoglobin = 1.34 .15 = 20.10 mL of O, per 100 mL of blood.

Hemoglobin forms a true chemical combination with O that depends on the partial tension of the latter gas. It is carried out according to the so-called Barcroft tension curve or oxyhemoglobinic dissociation. Oxygen in arterial blood is at a tension of 100 mm Hg. At the normal blood pressure of 100 mm Hg, 95% of hemoglobin is saturated in arterial blood.

In the capillaries and veins where the tension is 40 mm Hg, 75 to 85% of hemoglobin is saturated.

Since arterial blood contains 15 g of hemoglobin per 100 mL and only 95% is saturated (oxyhemoglobin) and the remaining 5% remains unsaturated, anoxic or reduced, it turns out that there is always 1 mL of oxygen left to combine in arterial blood: 15 g of Hb 1.34 mL = 20.10 mL per 100 mL of blood. The 5% unsaturated of 20.10 mL of O,, = 1 mL of oxygen to be combined. Added to this is the blood from the bronchial circulatory system, which, since it does not penetrate the minor circuit, is not oxygenated and contributes to maintaining arterial unsaturation.

Given the great diffusibility of the gases in both directions through the alveolar wall, the fact occurs that their concentration in the capillary is equal to that found in the alveolus, for which reason it is enough to measure the tension of CO in the alveolus to obtain a figure equal to the tension of said gas in the capillary. This tension is normally 40-43 mm Hg.

In summary, we can consider that the breathing mechanism requires for proper functioning:

1. That a minimum amount of O2 reaches the alveolus from the external environment.
2. That the transport of O from the alveolus to the tissues and that of CO from the tissues to the alveolus is adequate (external respiration).
3. That gas exchange between tissue cells and blood is correct (internal respiration).

EXPLORATION OF THE CHEST AND RESPIRATORY

9

LINES AND DEMARCATIONS FOR EXAMINATION OF THE CHEST AND THE TORIO BREATHING SYSTEM

It is advisable to remember the thoracic topography and point out, even briefly, the most important aspects.

When examining the chest, three parts are considered: anterior, posterior and lateral.

The elements to take into account in each part are:

Previous part

1. Supraclavicular fossa.
2. Infraclavicular fossa.
3. Intercostal region.
4. Mamelonar region.

Back

1. Superior or supraspinatus area (located above the spine of the scapula) that goes from the vertebral line to the scapular line.
2. External scapular zone, which continues the anterior one to the posterior axillary line.
3. The lower area, located below the line that passes under the shoulder blade and is called the base.

Side part

1. Upper area or axillary hollow above a horizontal line that passes through the mamelon.
2. Lower or subaxillary area.

The demarcations of the thorax to locate the underlying structures include the twelve thoracic vertebrae, twelve ribs on each side, the suprasternal fork, at the top of the sternum between the clavicles, and the sternal angle (of Louis), which is a slightly outward, palpable projection of the sternum, at the point of union of the manubrium with the body and where it articulates on each side, the second rib.

The angle of Louis is the starting point for counting the ribs and intercostal spaces. The latter have numbers that correspond to the number of the overlying rib.

Identification of the ribs by palpation is usually easier at the level of the midclavicular line than at the sternal border, where

the proximal sternal cartilages may interfere. Only the cartilages of the first seven ribs join directly to the sternum, directed from the outside to the inside and from the bottom to the top.

The costal margin refers to the proximal surface of the rib that descends and moves away from the sternum, and the costal angle is formed by the intersections of the costal margins.

Imaginary lines are also useful for determining the location of lung structures; They include the midsternal line, the midvertebral or midspinal line, the right and left midclavicular lines (vertical from the midpoint of each clavicle), and the anterior, middle and posterior axillary lines (right and left). The mid-axillary line descends vertically from the axillary dome.

The pulmonary apices extend in the anterior plane, approximately 5-6 cm above the clavicles. In the posterior plane, the apices extend to the first thoracic vertebra. The lower limits of the lung are located in the tenth thoracic vertebra (T-10) during expiration and in the twelfth (T-12) during deep inspiration.

The approximate location of the fissures that divide the lungs into lobes can be determined by observing the following lines of demarcation. Posteriorly, the lungs divide into the upper and lower lobes at an angle attached to the spinous process of T-3, obliquely downward and laterally. On the anterior surface, the lower lobe is divided from the upper lobe in the left lung, and from the middle lobe in the right, by a bilateral imaginary line that extends medially and inferiorly from the fifth rib, midaxillary line, to the sixth rib, midclavicular line. On the right lateral surface, the division of the right lobe and middle lobe is located by a line drawn medially from the fifth rib, midaxillary line, to the fourth rib, midclavicular line.

EXAMINATION OF THE CHEST AND THORIOUS

RESPIRATORY

The exam must be done with the person sitting. Even, if it is within our reach, we can use a rotating bench for these purposes, which allows rotation and facilitates successive exploration in various planes.

The chest must be naked, at least in the part to be examined, always respecting modesty. To do this, especially in the female sex, we can use a sheet, a towel, the person's own clothes, or any other fabric, to cover the planes that are not being examined at that time.

It is important to follow a sequential method so that we do not leave any areas unexamined.

In the order of execution, with respect to the regions to be explored, we can follow the following steps: first, the posterior plane is explored, from top to bottom, that is, from the regions of the vertices towards the lung bases; then we do it with the previous plane, in the same sequence; and finally, the lateral plane, for which the examined subject is asked to raise the ipsilateral arm and place the palm of his hand on the back of the neck or on the head or better yet, if the palm of the hand falls on the parietal region of the other side.

To examine the fissural region, the examined subject is instructed to cross the arm on the same side in front of him and place the palm of the hand on the supraclavicular region of the opposite shoulder. This exposes the interlobular fissure, since it coincides with the oblique position of the inner edge of the scapula.

In each plane, first one side must be examined, then the other and subsequently, a comparative exploration of symmetrical areas must be carried out.

In this way, after specifying the exploratory characteristics of each technique in one place, we immediately proceed to perform it in the symmetrical place, to establish the comparison. However, it is important to remember that there are physiological differences between the right and left sides that mean that in certain areas the normal findings are different on the right side compared to those on the left.

The four basic exploration techniques are used, following this rigorous order: inspection, palpation, percussion and

INSPECTION

Move around the subject to inspect the different regions and visible demarcation lines, in all three planes (posterior, anterior and lateral).

In the inspection of the chest, the condition of the skin, subcutaneous cellular tissue and musculoskeletal structures must be taken into account, just as in any other region of the body, as well as its configuration, which includes the shape and symmetry of the rib cage, the absence of doming or retraction in any hemithorax and the evaluation of the shape, direction and movements of the ribs and intercostal spaces.

Normal chest type

The shape of the thorax or thoracic configuration depends on the spine, sternum and ribs.

Compare the anteroposterior (AP) transverse diameter, looking at the thorax in profile, with the lateral transverse diameter, looking at it from the front, whose normal proportion

It is approximately 1:2. The AP diameter may increase in the elderly, as the dorsal curve of the spine increases, or its increase may be due to any age, musculoskeletal or chronic respiratory disorders (see Chapters 30 and 34).

Normally the thorax is symmetrical and is in relation to the constitutional type (normolinear, brevilinear or longilinear) of the examined subject, which gives it its own characteristics.

On the other hand, the rib cage is of regular shape, without vaulting or retractions, with the ribs and intercostal spaces oriented slightly downward in the posterolateral plane and without suction movements of the wall during inspiration (drawing) in the intercostal spaces, subcostal, suprasternal, supra or subclavicular regions.

The inspection in the particular examination of the respiratory system also includes the inspection of the trachea and the evaluation of respiratory movements.

When inspecting the anterior plane, note the position of the trachea, in the suprasternal hollow, above the fourchette, which should be in the midline, without lateral deviation to the right or left.

Study of respiratory movements

When we study respiratory movements, we must consider four fundamental aspects:

- Respiratory type.
- Frequency.
- Rhythm and depth.
- Ventilatory patterns.
- Thoracic breadth or expansion.

Respiratory type

With each breath, both hemithoraxes must have symmetrical and synchronous movements, up and down. With each effective inspiration, a downward movement of the diaphragm and an outward movement of the thorax and abdomen must occur. The opposite should occur with expiration. Women generally breathe with a chest movement, while men and children usually breathe with their diaphragm.

The normal respiratory type in women is upper costal. In the adolescent, in which the ribs are flexible, it is costal. In children and adults it is diaphragmatic or abdominal.

When there is asynchronous movement of the thorax and abdomen, in which during inspiration the abdomen moves inward and the thorax moves outward, ventilation is abnormal and ineffective.

Normal respiratory rate, rhythm and respiratory patterns

The evaluation of respiratory rate is carried out in practice when the rest of the vital signs are being evaluated: temperature, pulse and blood pressure, but if you are performing the particular examination of the respiratory system, you must keep in mind your examination (which tends to be forgotten), and perform it at the end or at the beginning of the inspection.

Respiratory rate examination technique

First of all, you should try to minimize the interference that could falsify the result. To do this, evaluate the respiratory rate (RR) while keeping your fingers over the site of the radial pulse, as if you were taking the pulse, because if the person notices that you are counting the respiratory rate, they may alter their ventilatory pattern, generally unconsciously.

Alternatively, if the individual is asleep, you can count the RR before assessing other vital signs or beginning the exam.

The second step is to observe respiratory movements. You can either visualize or feel the person's breathing movements. Visualization consists of observing how the chest rises and falls; Tactile observation consists of observing your hand placed gently on the subject's chest, which rises and falls along with the respiratory movements, which is not recommended, because it can create interferences, discussed above. Also observe the work of the respiratory muscles and the use or not of the accessory muscles.

Now count the respiratory rate. Use a clock with a secondary clock to count the number of times the chest rises and falls in 30 s and multiply it by two. If breathing is slow or irregular, count in one full minute. The normal adult respiratory rate is 12-20/min at rest.

Lastly, notice the rate and depth of your breaths.

Breathing should be calm and effortless. The time taken for expiration (E) is approximately twice the time for inspiration (1), therefore, the I:E time ratio is 1:2.

Don't forget to record your findings, especially noting the respiratory rate (RR).

Normal ventilatory patterns

- 15-20 breaths/minute.
- Regular, calm, effortless breathing, occasionally evident.
- Inspiration (time) ratio: expiration (1:E) 1:2.
- Average tidal volume (in adults): 350-500 mL.

Thoracic breadth or expansion

The thoracic amplitude should look the same in both hemithoraxes. Decreased amplitude in a hemithorax is abnormal. So if thoracic expansion is observed differently in one hemithorax in relation to the other, the abnormal finding is interpreted as decreased expansion in the hemithorax with lower amplitude, and not as increased thoracic expansion in the opposite hemithorax.

PALPATION

This examination complements the data obtained by the inspection and adds other aspects, such as sensitivity (pain), chest elasticity or expansibility and intensity of vocal vibrations or thrill.

Ask the subject beforehand if there is spontaneous pain in any part of the chest. Examine previously painful areas last. Use your fingertips to palpate the thoracic structures and intercostal spaces, looking for inflammation, asymmetry, bulging or retraction, and induced pain, and observe the quantity and quality of muscle mass on the chest wall. As you palpate, ask the person if they feel pain caused by the palpation being performed.

If there is a sinus or fistulous tract, palpate it.
Also palpate around any wounds or connections from invasive procedures, such as endotracheal tubes or intravenous catheters, to look for crepitus.

The sternum, costal cartilages, ribs, intercostal spaces and spine should not be painful to palpation. Normally, the muscles feel smooth and symmetrical. Crepitation is an abnormal canglorous sound produced when air contained in the subcutaneous cellular tissue is palpated.

Thoracic expandability or elasticity

Examination of thoracic expansibility by palpation can be performed with a posterior approach or an anterior approach. Usually only the posterior approach is used, or both are used in this order.

Posterior approach

A. Base maneuver (fig. 9. 1).

Place your hands on the posterolateral region of the chest as if gripping it, with your thumbs at the level of the tenth rib, pointing toward the spine.
Pull the skin slightly towards the center with your hands, trying to bring both thumbs closer together, so that the sides form an open downward angle.
Ask the person to breathe deeply and observe the movement of their hands, the opening of the angle and the separation of their thumbs, which are produced by thoracic expansibility at the base.

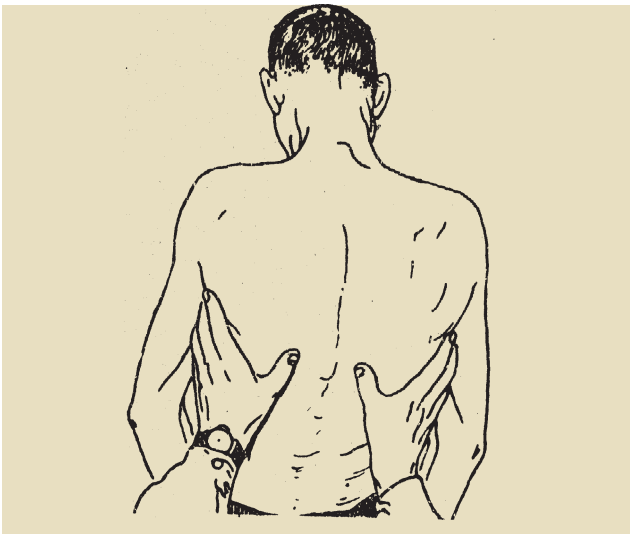


Fig. 9.1 Exploration of the expansion of the bases, from the posterior plane.

B. Vertex maneuver.

Place both hands on the examinee's shoulders, with your thumbs at the level of the first rib, pointing toward the spine. Pull the skin slightly towards the center with your hands, to try to bring both thumbs closer together, so that they form the sides of an open angle downwards. Ask the person to breathe deeply and observe in their hands the same thing they observed with the base maneuver. and thus evaluate the thoracic expansibility at the level of the vertices.

Previous approach

Place your hands in the anterolateral region of each hemithorax, with the thumbs directed towards the sternum, extended along the costal margin, which meet in the anterior midline, at the level of the sixth chondrocostal joint, and the other fingers, directed horizontally outwards, reach below the axilla to the mid-axillary line (fig. 9.2).

Ask the person again to breathe deeply and observe the movement of their hands, the opening of the angle and the separation of their thumbs, produced by the lower thoracic or base expandability.

Normally, when the person takes a deep breath, the thumbs should be separated from the spine an equal distance on each side.

Physiologically, elasticity is greater in the child, lower in the elderly and intermediate in the adult, which explains the difference in thoracic expansion in each one.

A decrease in thoracic expansibility in a hemithorax may indicate pulmonary or pleural disease (see Chapter 34).

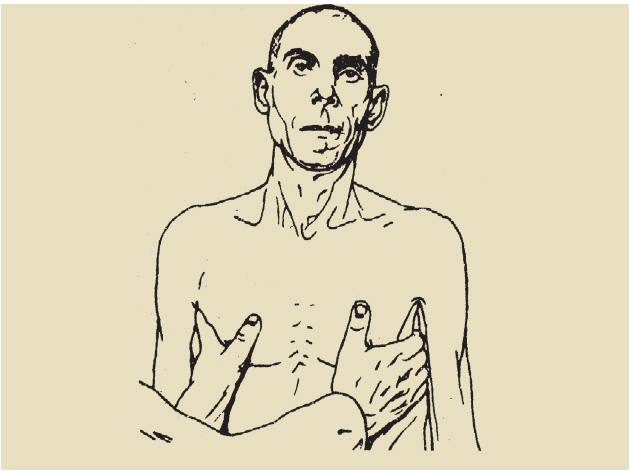


Fig. 9.2 Exploration of the expansion of the bases, from the previous plane.

Fremitus or vocal vibrations

Origin and transmission

Vocal vibrations originate in the vocal cords during phonation and are transmitted through the air column of the tracheobronchial tree to the lung. These are free vibrations originated by an elastic body that has its own frequency, the vocal cords, and that make the lung vibrate with forced vibrations that reach it. The forced vibrations of the lung are transmitted to the pleura and chest wall, where we palpate them.

From physics we know that the conduction of a sound vibration will be greater, the closer the frequency of the sound is to that of the conducting body. So you-

Let's say:

- Normal lung: 90-130 vibrations per second.

Man's voice: 100-130 vibrations per second.

- Woman's voice: 260 vibrations per second.

More than 500 vibrations per second are not transmitted by the lung.

Vocal vibrations are attenuated before reaching the chest wall due to the reflection that sound waves suffer when passing through media of different densities.

It has been rightly said that vocal vibrations "run well, fly badly and swim worse"; In the classes we teach to students, we use the following simile: if we apply this aphorism to the chicken, we will see that this animal can run relatively fast, however, it only flies at low altitude and short distances and, of course, dies if it submerges in water. In this way it is easier to remember the concepts that physiology and physics teach us in relation to vocal vibrations.

Physiological variations of vocal vibrations

Vocal vibrations are modified by: age, sex, voice and topography.

Age. Vocal vibrations in children are less intense, because the voice is high-pitched and less strong; In the elderly, vocal vibrations are less intense because the voice is weak and veiled, and in adults they are more intense because the voice is deeper and stronger.

Sex. In women, vocal vibrations are less intense, because the voice is high-pitched and less strong.

Voice. Vocal vibrations are in direct proportion to the intensity and inversely to the tone; There is the so-called high or head voice and the deep or chest voice. The vibrations are greater in the upper part of the chest in people with high voices and more intense in the lower part in people with deep voices.

Topography. The topographic location of vocal vibrations is variable. The thicker the wall, the fewer vibrations reach the palpating hand, and vice versa.

According to the Monneret scale, vocal vibrations decrease in intensity in this order:

1. Larynx.
2. Trachea.
3. Last cervical vertebrae.
4. Infraclavicular region.
5. Posterior and lateral regions of the thorax.
6. Sternum.
7. Supraspinous fossa.

The range of vibrations per second can be established according to the scale seen in figure 9.3.

Vocal vibration exploration technique

Perform the examination in all three planes, starting from the posterior and lateral, from top to bottom in one hemithorax, then in the other and. finally, comparative palpation.

1. Stand behind the subject and ask the person to say “thirty-three,” each time they feel the palpating hand.

   Usually a word is used, usually a number, whose consonants produce sufficient vibration of the vocal folds. In some Anglo-Saxon countries "nighty nine" (99) is used and in the Arabs 'arbaa arbaín (44).

2. Rest your hand on the right hemithorax, flat, parallel to the transverse axis, so that the palmar region of your joined fingers rests on the intercostal spaces and the ribs, starting from above down through the suprascapular region; Bordering the scapula, move to the interscapulovertebral region and then to the entire base, including the lateral region, while ordering the expression of the number with a "say" in each position.

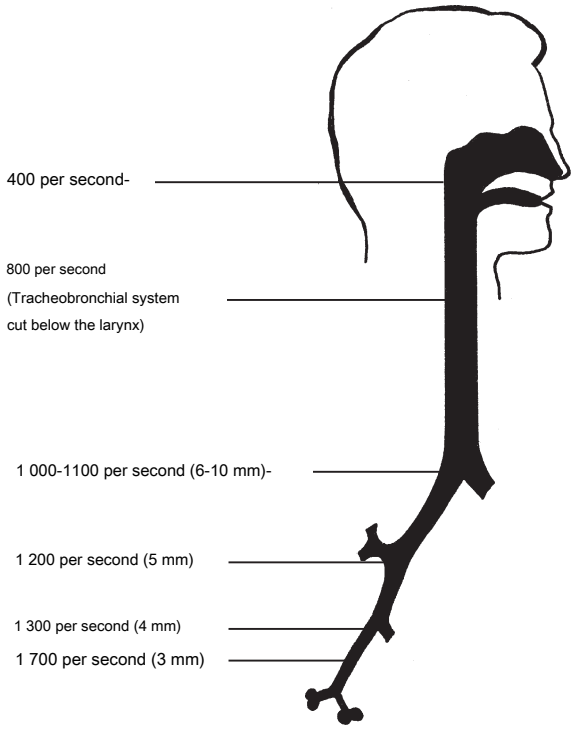

400 per second-
800 per second
(Tracheobronchial system
cut below the larynx)
1 000-1100 per second (6-10 mm)-
1 200 per second (5 mm)
1 300 per second (4 mm)
1 700 per second (3 mm)

Fig. 9.3 Schematic longitudinal section of the bronchial tree with the frequency of free vibrations of the component tubes, according to Martini and Müller.

In some Anglo-Saxon countries it is used to place the ulnar edge of the hand over the intercostal spaces, instead of palmar palpation, which has the advantage of feeling the vibrations without interference from the ribs, but the edge of the hand is less sensitive and, in our opinion, it is preferable to use the palmar region of the fingers.

3. Repeat the same on the left hemithorax.

4. Once you have the idea of the variations of vocal vibrations in each hemithorax, whether physiological or pathological, perform comparative palpation, with the same path from top to bottom, but passing in each position, from one hemithorax to the symmetrical position of the other, so that you can compare the palpatory sensations.

If after concluding the comparative technique, you still have doubts about the evaluation of a certain place, repeat the comparison of that site as many times as necessary, until you are sure of the result of the evaluation.

5. Explore vocal vibrations in the anterior plane, mainly in the infraclavicular regions, and complete the exploration of the lateral planes.

It is advisable to explain well to the subject, not only what the technique consists of, but also the seriousness of the test, due to its usefulness, and the need for him to express only the number and with the same intensity, each time he is asked.

6. If you do not find any evidence of alteration, record in writing: "vocal vibrations (or VV): preserved."

PERCUSSION

Sensations provided by percussion

Digit-digital percussion of the chest produces two types of sensations:

1. The auditory, which is due to the loudness of the lung.
2. The tactile, which is due to the elasticity of the lung.

Tactile sensation is a resistance to the finger, which increases as the normal percussive sound decreases.

Sometimes the sensation of resistance and the percussive sound are increased. This occurs in rare cases of pulmonary emphysema, in which there is premature ossification of the costal cartilages and rigidity of the skeleton.

In these cases, the aeration of the emphysema increases the percussive sound and the ossification and rigidity of the skeleton increase the tactile resistance.

The most used percussion is the digit-digital, already studied in Chapter 2.

Sounds obtained by percussion of the chest. physical characters. Origin of clear lung sound and other sounds

Sound is a form of vibratory energy generated and conducted by elastic bodies. Sound bodies produce longitudinal waves in the air that, when acting on the ear, determine the sound sensation. The ear perceives sounds of 16,000-40,000 vibrations per second.

The fundamental characteristics of sound are: intensity, tone, timbre and duration.

The intensity depends on the amplitude of the vibrations.

The tone or height depends on the frequency, with the highest frequency being the high tone, and the lowest being the low tone.

The timbre depends on the nature of the vibrating body.

The sound is produced by the fundamental tone and the higher harmonic overtones, of different intensity and tonality than the fundamental sound. When the overtones are many and of great intensity, they hide the fundamental sound, producing what is called noise.

The clear lung sound is caused by: the vibration of the aerated lung parenchyma (fundamental cause) and the rib cage (resonator).

The greater or lesser vibration of the lung depends on three causes: the volume of the vibrating tissue, the density and the tension.

The greater the volume of the vibrating tissue, the characteristics of the sound will be: greater intensity, long duration and low tone.

The greater the density of the tissue, the sound will be: lower intensity, short or short duration and high or sharp tone.

The greater the tension of the tissue, the sound will be: lower intensity, short or short duration and high or sharp tone.

Depth reached by percussion

The percussion blow vibrates the entire lung, but the percussive noise is caused by the vibration of the percussed area, equivalent to a hemisphere with a radius no greater than 6 cm from the skin surface or 4 cm from the surface of the lung. This radius is further limited when hitting a rib, which is why it is preferable to hit the intercostal spaces.

For an injury to modify percussion, it must be superficial and of sufficient volume.

Physiological and topographical modifications of percussive sound

The common cause of physiological modifications is the greater or lesser thickness of the wall. Furthermore, the percussive sound changes with the breathing times, being hyperresonant on inspiration and hyposonorous on expiration.

The topographic modifications are due to the unequal distribution of muscle masses and the relationship of the lung with more or less hard neighboring organs.

According to these questions we will have:

- Maximum loudness: infraclavicular and axillary regions.
- Minimum loudness: supraspinous regions.
- Medium loudness: infrascapular regions.

Loudness in the previous plane

First and second spaces: greater sound.

Second and third spaces in women: submate or mate, due to the presence of the breasts.

Third rib and third left space: submate, due to the presence of the heart.

Fourth and fifth right spaces: submate, due to the presence of the

Left costal margin: hypersonic, due to the presence of Traube's semilunar space (stomach).

Loudness in the rear plane

In general, the loudness is lower than in the previous plane.

Scapular region: the least loudness.

Interscapulovertebral region: louder.

Infrascapular region: maximum loudness.

Right eighth intercostal space: submate or matte, due to the presence of the liver.

Loudness in the lateral plane

The sound here is intense. On the right side it decreases downwards through the liver and on the left side it becomes tympanic due to the presence of the stomach and the splenic flexure of the colon.

Percussion of the bones of the thorax

Clavicle. Sonora, except in the external third due to the presence of shoulder muscle masses.

Breastbone. Sound in the handlebars and body. Mate in the xiphoid appendix, due to the presence of the liver.

Backbone. Sound from cervical vertebra VII to dorsal XI. It has value in pleurisies, because it disappears giving a dull sound.

In the scoliotic spine, two areas of submattitude appear opposite the lateral convexity of the spine.

Technique for examining pulmonary percussive sound

Perform percussion of the thorax by planes, starting with the posterior plane and following the same steps and the same route explained for the palpation of vocal vibrations; but this time, placing the pleximeter finger over the intercostal spaces.

To evaluate pulmonary percussion sound, the ribs, scapulae, or any other bone structure are not percussed.

Before starting to strike the posterior plane, ask the person to cross their arms over the chest, or have each arm cross the midline, directing each hand towards the opposite knee, with the aim of moving the scapulae outwards and leaving more space exposed for striking.

Remember that in each plane, first one hemithorax is percussed, then the other and, finally, the comparative percussion is

In the posterior plane it is also important to percuss the thorax to determine the diaphragmatic excursion.

Diaphragmatic excursion is the distance between the levels of dullness with deep inspiration and with full expiration. To do this, ask the subject to take a deep breath and locate and mark with a dermatographic pencil the limit of diaphragmatic excursion, when the lung resonance changes to dullness.

Next, ask the person to perform a forced expiration to again determine the limit of thoracic excursion. Now measure the distance between the two boundaries, which normally has a range of 3-6 cm. The diaphragm should be slightly higher in the right hemithorax, due to the position of the liver; so the extent of your incursion will be slightly less on the right side.

In the anterior plane, the percussion is completed with special techniques, when the cardiac area, liver and spleen are explored.

When no lung sound abnormalities are detected, record the percussion result as: normal lung sound.

AUSCULTATION

To obtain the maximum benefit from this technique, it is cardinal to meet some requirements in its implementation. With a view to this, the following aspects must be taken into account: the place where the procedure is going to be carried out, the positions and attitudes of the doctor and the subject being examined, the characteristics inherent to the stethoscope and its use, and finally, the execution of the auscultation as such.

The place where the auscultation is going to be carried out should ideally have a series of attributes: privacy, a calm, quiet environment, with a pleasant temperature, safe from sudden changes in temperature and outside noises.

For his part, the doctor will try to adopt a comfortable position and, above all, avoid leaving your head in a forced position. When the head is too low, ringing or dullness of the ear usually occurs, making auscultation difficult. You must perform the technique calmly and put all your attention on what you are doing.

The examinee will be placed in the most comfortable and, at the same time, convenient position to meet the objectives of the auscultation.

Auscultation of the respiratory system can be done with the subject sitting, standing, or lying down when dealing with a bedridden patient, but the objectives are optimally achieved with the person sitting. It must be done without interposing clothing. It is a mistake to auscultate over clothing, much more so if the fabric is silk, because auscultation is hindered and accessory noises can be created that can be confused with abnormal respiratory sounds.

In the few cases in which immediate, direct auscultation is going to be used (practically obsolete), a fine linen cloth can be inserted, for obvious hygiene reasons.

We must ask the subject to be as relaxed as possible and to position himself as follows: arms hanging along the chest, forearms resting gently on the thighs, and head and shoulders slightly inclined forward.

The pleasant and stable temperature, together with muscle relaxation, prevent the appearance of involuntary muscle contractions that can distort auscultatory findings.

The examinee should be invited to breathe calmly and regularly, without effort, but more deeply than usual. Let him breathe through his nose with his mouth half open or through his mouth, according to his preference or habit, always taking care not to make nasal or mouth noises.
Many times it is appropriate to show the subject how to breathe by doing it ourselves so that he can see us. Breaths should try to be equal, except when we tell you to take a deeper breath, or to talk, or to cough.

It is important to remember, regarding the auscultatory technique, to follow the same sequential and orderly method that was explained at the beginning of the chapter, for the entire exam.

Auscultation of the bedridden patient will be described in the Propedeutics Section.

Currently, with few exceptions, the auscultation used is mediate, with the use of a binaural stethoscope. Since most respiratory sounds are high pitched, the closed diaphragm type receiver (Bowles capsule) is the most used for respiratory aucultation.

Care must be taken to ensure that the diaphragm is placed firmly and completely on the skin. If the receiver is not fully inserted, it can cause accessory noises that sometimes resemble rales (pseudorattles).
On the other hand, if excessive pressure is applied, it causes dilation of the skin, which in turn becomes a type of diaphragm and causes the exclusion of low-pitched sounds.

In very narrow intercostal spaces or in the supraclavicular fossae, the bell receiver (Ford type model) can be used, taking care to press on the skin enough (but not excessively) so that it becomes a diaphragm. This bell receiver is not very suitable for very thin people, with protruding ribs and very sunken intercostal spaces, nor for auscultating the posterior plane of a person in passive recumbency.

The stethoscope earpieces should fit snugly, with moderate pressure. The eartips must be clean, comfortable and adapt well to the ear holes and prevent sound "leaks". We must avoid any rubbing of the instrument with our hands or clothing or that of the subject being examined, as well as crossing the rubber tubes or breathing heavily on them.

The passage of the diaphragm over the chest hair can cause sounds similar to rales. This can be avoided by wetting the hair.

Keeping the aforementioned aspects in mind and while the person being examined breathes regularly, focus your attention on normal respiratory sounds, evaluating the main elements of the vesicular murmur such as: rhythm, intensity, tone and timbre.

After clearly specifying the characteristics of the vesicular murmur, one must proceed to the identification of adventitious noises, such as rales, modifications with cough, and auscultation of the natural and whispered voice, which will be discussed in the Propedeutics Section.

Normal breath sounds

- Glottal murmur.
- Vesicular murmur.
- Bronchovesicular respiration.

Glottal murmur

Also called laryngotracheal noise, tracheal or bronchial breathing.

Audible. Below the cricoid cartilage.

physical characters. Intense, inspiratory and expiratory noise, similar to blowing through a medium-caliber tube and is imitated by breathing hard with the mouth half open, placing the tongue on the palatal vault. It is an intense noise, with a high pitch (more on expiration), with a tubular timbre, inspiration and expiration separated by a small silence, the expiration being more intense and lasting.

Location. At the level of the larynx and trachea it is called strong bronchial or tracheal breathing. In the lower half of the trachea and main bronchus and in the second right intercostal space next to the sternum, it is called bronchial breathing of moderate intensity. At the level of the fourth dorsal vertebra, this glottic murmur is called bronchovesicular respiration.

Vesicular murmur

Also called Laennec's respiratory sound or vesicular respiration.

Audible. In the infraaxillary, infrascapular and infraclavicular regions; in the latter, especially in the first two spaces towards the outside.

physical characters. Intensity: lower than in the glottal murmur; tone: serious; duration: inspiration and first part of expiration.

This noise is similar to that produced by a bellows whose valve makes no noise, or to that made by a man who in a quiet dream takes a deep breath, or to the noise made by the breeze through the foliage of a forest.

It is imitated by inhaling air through the mouth, with the lips in position to pronounce the Vo la FE.

The inspiratory tone is similar to the note “D” of the free string of the violin and the expiratory tone is similar to that of the note “C” of the free string of the violin, that is, a lower tone.

bronchovesicular respiration

Intermediate between the previous two.

Audible. Where the bronchi project, as occurs at the apex of the right lung, due to the greater proximity of the trachea at that level.

Topographic distribution and physiological variations of normal respiratory sounds

The intensity of the glottal murmur has been referred to previously. We will now address, in more detail, the study of the vesicular murmur.

The vesicular murmur is more intense in the right hemithorax because the caliber of the bronchus on this side is larger. Studying its intensity by region, we found that in the anterior plane it is greater in the infraclavicular region, that is, in the first two intercostal spaces. In the axillary plane it is greater above than in the infraaxillary region. In the posterior plane it is greater in the interscapulovertebral region, less intense in the infrascapular, even less in the suprascapular and, finally, minimal in the scapular region, due to the presence of the bone sheet of the scapula covered with muscle, which is why it is not heard over it.

The vesicular murmur is more intense the less thickness and more elasticity the thorax has.

Modifications due to age. In children, the vesicular murmur is intense and acute (in Faspirada), which is why the supplementary breathing is called puerile, due to its similarity to the breathing of the child.
In the elderly, expiration is lengthened, which is called emphysematous respiration.

Modifications by sex. In women, breathing is less intense, the vesicular murmur is heard more intense and high-pitched in the upper portion of the thorax due to the type of upper costal breathing typical of their sex.

Auscultation of cough and normal and whispered voices will be described in the Propedeutics Section, in Chapter 34.

10

CLINICAL ANATOMY AND PHYSIOLOGY OF THE SYSTEM

CARDIOVASCULAR

CLINICAL ANATOMY

The complex cardiovascular system's function is to originate and maintain blood circulation. It is made up of a central organ, the heart, which acts as a suction and delivery pump and is responsible for creating the necessary impulse for the blood, through an adequate distribution system, to irrigate the tissues.

The heart is located asymmetrically in the thorax, with two thirds to the left of the midline and one third to the right; hence its anatomical axis deviates 45° in the normolinear subject, less in the longilinear and more in the brevilinear subject.

This organ is made up of two parts: the right heart and the left, which function simultaneously and are connected to two different distribution systems. The right heart sends its blood through the pulmonary arteries to the vascular network of the lung, which is called the minor circulation. The left heart sends its blood through the aorta artery to the rest of the body, which is called the greater circulation.

Through the venous system, return blood circulates to the right heart where it is poured; For this there are two large venous trunks: the superior vena cava and the inferior vena cava. Return blood from the lungs reaches the left heart through four pulmonary veins.

The blood that reaches the halves of the heart in this way does not pass directly to the two large cardiac cavities called ventricles, but rather first fills the atria or atria, so each cardiac half is divided into two chambers: atrium and ventricle.

The vascular compartments that make up the body have special characteristics:

a) Hepatic system. It is made up of the hepatic artery, which carries a small part of the blood to the liver, and the portal system (portal vein, sinusoids and suprahepatic veins), which carries most of the blood to this organ and drains it towards the inferior vena cava.

b) Gastrosplenointestinal system. Here the blood flows to the portal vein and from this to the liver.

c) Inferior cava system: Both the portal system and the independent systems of the kidney, genital organs and extremities

inferior veins empty into a large venous trunk called the inferior vena cava.

d) Azygos system. Another peculiarity of the venous system is the following: the veins of the thoracic walls and the intercostal veins are afferent to a large collecting duct, which is the azygos vein; This, at the level of the abdomen, connects with the inferior vena cava system, then crosses the thorax to the right of the vertebral column and, finally, empties into the superior vena cava. Circulation disorders of the azygos vein usually cause harmful effects in the pleural cavity (effusions, etc.).

e) Upper cava system. The cerebral vessels, those of the neck and those of the upper extremities, also empty into the superior vena cava.

f) Arterial system. All vessels that are in some way related to the ventricles are called arteries, regardless of the type of blood that circulates through them, since the name itself is determined by the characteristics of its histological structure. The large arteries are successively subdivided into smaller ones until they constitute microscopic vessels of different structure called capillaries. From the capillaries, the blood passes into small veins that flow into larger veins until they form the large venous trunks that carry the blood back to the heart.

g) Lymphatic system. It represents an accessory route through which fluids from the interstitial spaces can reach the blood.

The lymph is collected by the lymphatic vessels and first passes through the nodes; From these, the lymph is taken by new collectors and is ultimately poured into two thick trunks, which are: the thoracic duct on the left, and the great lymphatic vein on the right, which lead the lymph to thick veins at the base of the neck (confluent between the internal jugular and subclavian veins).

heart anatomy

The heart is considered to have two serous membranes: the endocardium, a membrane that covers the entire internal surface of the organ and forms the valves, and the pericardium, a type of sac that contains the heart. Between the two serosae is the muscular layer or myocardium (Fig. 10.1).

The heart is placed obliquely in the tőrax.

with the base backwards and to the right, the vertex forwards and to the left. This characteristic determines that the projection of this organ on the anterior face of the thorax or precordial region is formed by the right ventricle for the most part, and only a small part (the left) is made up of the left ventricle; back and to the right corresponds to the right atrium. The posterior surface of the heart is made up of the left ventricle and mainly the left atrium. Its diaphragmatic surface is formed by the left ventricle, the right ventricle and the right atrium, and, finally, the base corresponds to the two atria.

This organ is made up of four cavities or chambers: two atria and two ventricles that can be specified externally by the existence of fissures or grooves where its nutritional vessels are located. These grooves are: the atrioventricular and the two intertricular grooves (anterior and posterior).

Right heart

The right atrium, with a fairly thin muscular wall, performs a very modest contractile function; It has a triangular evagination in its anterosuperior part: the right appendage. It receives venous blood from the superior and inferior vena cava and the coronary sinus. It is separated from the right ventricle - with which it continues - by means of the atrioventricular valve, called tricuspid, because it has three leaves or leaflets, and from the left atrium, by the interatrial septum or septum interauricula, 2.5 mm thick and made up mostly of muscle tissue, although its posteroinferior portion is thinner and fibrous in nature (foramen ovale).

This atrium is located at the base of the heart towards the back and to the right. It ends with: the superior vena cava, which flows into a kind of sinus or excavation isolated from the rest of the cavity by a narrow groove or slit; the inferior vena cava, which has a type of valve called the incomplete semilunar valve or Eustachian valve, which partially prevents blood reflux (the orifices of both vena cavae are located near the interatrial septum); the coronary veins, which empty through the coronary sinus; the Thebesian valve, which is located at that level, near the lower edge of the interatrial septum.

The right atrioventricular septal defect is located along a horizontal line that runs from back to front, from right to left and slightly downward;

This hole is oval in shape, measures about 12 mm and has a circumference of approximately 100-120 mm. It is closed by the tricuspid valve, each of whose three leaves or leaflets is joined to the ventricle by a chorda tendineae that is inserted into the muscles called papillaries. The different valve leaves are arranged as follows: a lower one, near the diaphragm; another medial, next to the interventricular septum and the third, anterior, in front of the orifice of the pulmonary artery. Each papillary muscle controls the adjacent halves of two valve leaflets.

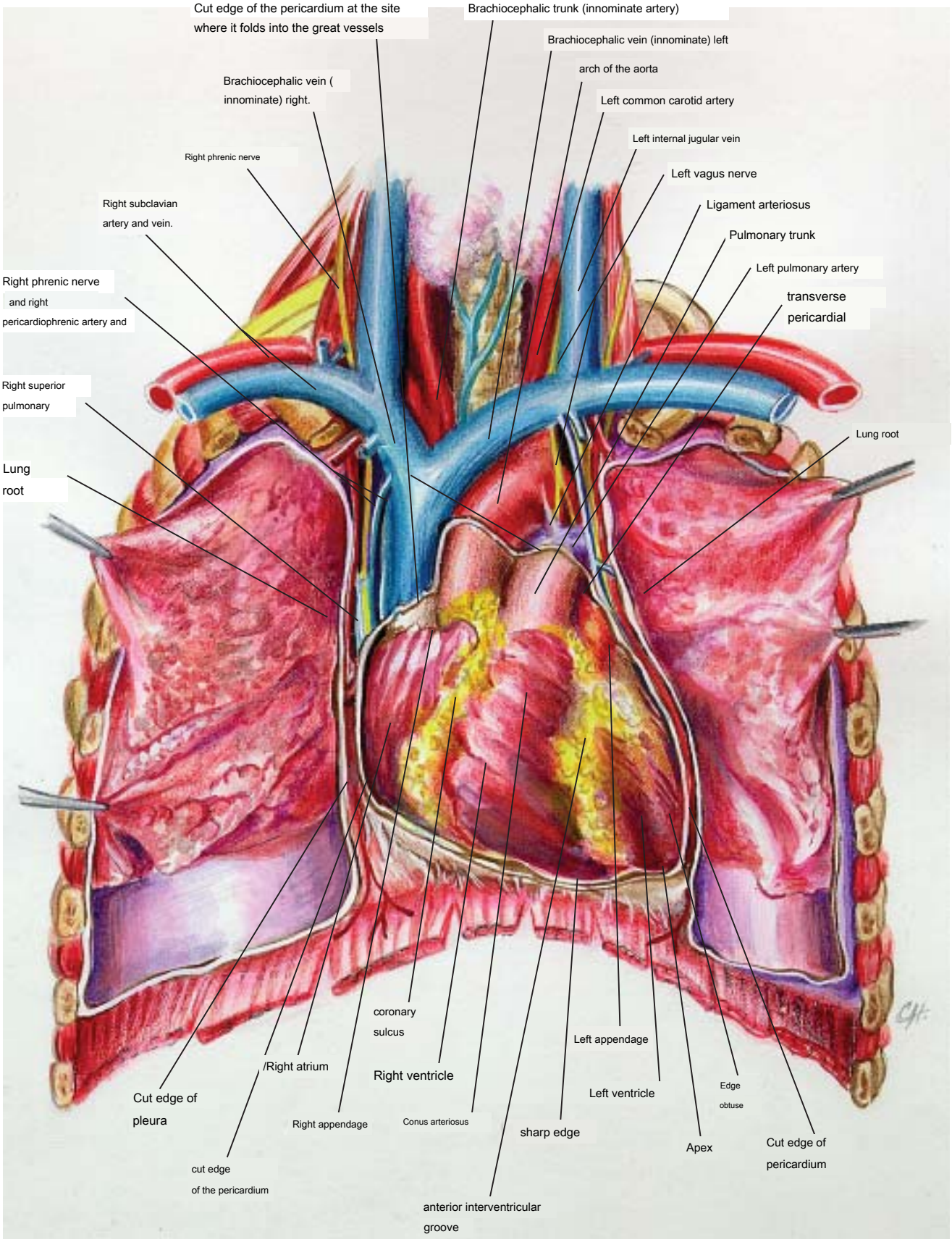


Fig. 10.1 Anatomy of the heart.

The right ventricle receives blood from the atrium through the tricuspid valve. It consists of two different parts: one is the inlet tract or filling chamber, located below the tricuspid orifice; the other is the outflow tract, which is an oblique infundibulum upwards and to the left, and which constitutes the passage between the filling chamber and the orifice of the pulmonary artery; Both parts are separated from each other by a slightly narrow area, the ostium infundibuli or door of the infundibulum.
On the wall of the filling chamber, and especially in the septum, there are prominent muscular columns; On the contrary, the infundibular wall is smooth.

When making a cross section of the right ventricle, its circular shape and its wall are observed to be approximately 4.1 mm thick at the base; the pulmonary artery, which heads upward and then to the left, whose circumference is approximately 70 mm in diameter and has a pulmonary sigmoid valve with three leaflets, each with a small nodule towards the middle part of its free edge. The insertion of the lung, in the form of fibrous tissue, is lost between the muscle fibers of the ventricle. Above each valve there is a kind of dilation niche called the sinus of Valsalva.

left heart

The left atrium is smaller than the right and has, like the left atrium, an outpouch: the appendage.
It receives blood from the lung through the pulmonary veins on its posterior surface, two of them near the interatrial septum and the other two far to the left. A small groove separates this structure from the left ventricle.

This atrium is the most distant cavity of all the

that make up the cardiac organ, and that is why it comes into intimate contact with the descending aorta, the esophagus and the spinal column. It continues in communication with the corresponding ventricle through the left atrioventricular valve or mitral valve.

The mitral valve is rounded in shape, with a circumference of approximately 90-101 mm; It is directed obliquely forward to the left and somewhat downward. It is made up of fibrous tissue that forms two valves, which is why it is also called bicuspid.

The valves have, like those of the tricuspid, chordae tendineae and papillary muscles; One of the leaflets is arterial and medial, which is why it is called the mitral aortic leaflet, and the other is located laterally and posteriorly. Each papillary muscle, one anteroleft and one posteroright, controls the adjacent halves of each leaflet.

The left ventricle receives blood from the atrium through the mitral valve and sends it to the aorta through the semilunar valves. Here it happens the same as on the right side; The blood mass is divided into two well-defined currents: the inlet, located posteriorly, and the outlet, located in an anterior plane. The ventricle is circular in shape, with a thickness of its walls of approximately 10.2 mm.

The aorta at birth is directed obliquely upward, backward and to the right. Its lumen is rounded and about 67 mm in circumference. It is separated from the ventricle by the aortic signoidal valve, which has three leaflets, similar in every way to those of the pulmonary artery.

The interventricular septum is formed by muscle fibers whose thickness is approximately 10 mm, although at the base it is made up of a thinner fibrous tissue (2 mm) called pars gallinacea, triangular in shape, with a concavity to the left.

It should be noted that the anterior leaflet of the mitral valve is inserted on the septum, a little higher than the septal leaflet of the tricuspid, in such a way that in an extension of a few millimeters the septum directly separates the left ventricle from the left atrium.

Structure of the myocardium

The muscular tissue of the heart, myocardium, despite having a striated appearance, differs from skeletal muscles by being composed not of isolated fascicles, but of a network of muscle fibers linked together and by its central nucleus. The myocardial fiber is particularly rich in mitochondria, which shows the high oxidative metabolic level of these fibers.

Structure of the atria

Two muscular layers are distinguished in the atria:
one superficial and one deep. The superficial one is made up of fibers arranged circularly or transversely, and the deep one, of longitudinal fibers that, starting at their ends in the fibrous rings that surround the atrioventricular orifices, cover each atrium in the shape of a loop. The fibers of the superficial layer cover both atria; those of the deep layer belong separately to one or the other atrium. Strong circular fibers are found around the large venous vessels that empty into the atria.

It has been noted that the atrial myocardium has a greater water content and collagen fibers than the ventricular one.

Structure of the ventricles

According to an old description, the ventricles constitute two muscular sacs wrapped within a third. More recent studies show that the myocardium is made up of numerous muscles that can be dissociated and that have independent blood supply.

In the ventricles three muscular layers can be distinguished: a thin superficial layer (external layer), formed by longitudinal fibers that begin in the fibrous lamina of the right atrioventricular sulcus and are directed obliquely downwards, also extending to the left ventricle and after surrounding the tip of the heart, they coil like a handle towards the interior, constituting the internal longitudinal layer, whose fibers are inserted at their upper ends into the rings. fibrous.
The middle layer, located between the longitudinal, external and internal layers, whose fibers follow a more or less circular direction, with the particularity that, unlike the fibers of the superficial layer, they do not pass from one ventricle to the other, but are independent for each of them.

Myocardial fibers (myofibrils) constitute a special class of striated muscle fibers, and their special character is the property of subdividing and uniting repeatedly, in such a way that they constitute a kind of network or syncytium.

Automatic or specific heart system

Numerous formations of more specific tissue are found at the level of the myocardium, which have a highly developed ability to produce periodic stimuli and to coordinate the activity of the muscles of the different chambers. This property is called automatism. The automatic system, also known as the excitoconductive system of the heart, can be divided into two parts: the sinoatrial system and the atrioventricular system.

Sinoatrial system

It is made up of the Keith-Flack node, sinus node or Sinu-Atrial node (S-A) and its relationship fibers.

The S-A node is located at the junction of the superior vena cava with the right atrium and extends in the form of a curvilinear ridge downward, to the mouth of the inferior vena cava. It has an approximate length of 25-30 mm and a thickness of 2-5 mm.

The connecting fibers extend from the node outward in a radiating manner in all directions, and quickly submerge or anastomose with the fibers common to the atrium.

There are no anatomically differentiated branches in the atrium. Despite this, certain branches draw attention due to their short size. One of these is Bachmann's, sinoatrial fasciculus, which connects the left atrium with the right; Other arborizations connect the S-A node with the atrioventricular node (A-V node). This conductive system is composed made by a complex of muscle fibers with a special structure (internodal bundles) that are distinguished by their scarcity of myofibrils and abundance of sarcoplasm, which is why they are lighter.

atrioventricular system

It is formed by the A-V node with its relationship fibers, the bundle or fascicle of His and its branches or Purkinje network.

The A-V node (Tawara node) is a rounded formation that is present in the posterior and inferior portion of the atrial septum. It is about 5 mm long by 2 or 3 mm wide; The relationship fibers connect it with the right atrium, with the septum and with the left atrium. Gradually this nodule becomes a fine branch that is the bundle of His or atrioventricular fasciculus, which is directed through the fibrous tissues to the base of the ventricles.

The bundle of His divides into two branches: right and left, and the latter is subdivided into the anterior and posterior bundles.

In a certain sense, the left ventricle has two Purkinje networks, one dependent on the anterior division, and the other on the posterior division. The two divisions of the left branch insert respectively into the anterior and posterior papillary muscles of the left ventricle. The profuse arborization of the Purkinje networks only occurs after the two divisions reach their respective papillary muscles.

The right branch is thinner and longer than the trunk of the left branch, and inserts into the anterior papillary muscle of the right ventricle.

The segment of the bundle of His from which the left branch arises is called the branching portion, while the segment that remains above it is the penetrating portion. There is no true bifurcation of the bundle of His in humans. The terminal end, where the right branch separates from the most anterior part of the left branch, has been called a pseudobifurcation.

In normal human beings the bundle of His represents the only muscular connection between the atria and ventricles.

The conducting tissue of the heart is slightly different from the rest of the myocardium. Its fibers are finer, less clearly striated and mixed with a system rich in nerve cells, nerve trunks and nerve fibrils.

The presence of nervous elements has been described in the fascicles that are part of the general nervous system of the heart, therefore, the sinoatrial and atrioventricular fascicles must be considered as complex neuromuscular formations.

Structure of the pericardium

The visceral pericardium is a thin sheet of cells attached to the heart, which is called the epicardium. The parietal pericardium, on the other hand, has a serous tunic supported by a strong fibrous tissue that forms a kind of sac. The mediastinal pleura largely covers the pericardial sac. A discrete amount of fluid is normally present in the pericardial cavity.

This facilitates the movements of the heart.

The base of the pericardial sac adheres intimately to the diaphragm, forming a triangular area that includes a fibrous center and part of the diaphragmatic muscle. A thin hiatus (Larray's space) allows the pericardium to be in contact with the peritoneum.

The pericardium has some thickenings called ligaments that connect the sac with neighboring structures. The upper part of the pericardial sac remains in intimate contact with the great vessels, mainly with the superior vena cava, the aorta and the pulmonary artery; At this level the fibrous tissue of the pericardium gradually fuses with the external tunic of the vessels. On the contrary, the serous tunic of the sac is continuous with the epicardium. The line of insertion is slightly irregular and is approximately 50 mm in circumference, beginning at the origin of the pulmonary artery and descending to approximately 68 mm from the aorta.

The superior vena cava has an extrapericardial section and another intrapericardial section. The inferior vena cava only has a short intrathoracic course (10-15 mm).

The opening of the two hepatic veins inside the cava also occurs at the level of the diaphragm or slightly above it; In such cases the pericardial covering covers not only part of the inferior vena cava, but also a small portion of the hepatic veins.

(fig. 10.2).

Structure of the endocardium

The endocardium is a whitish, translucent membrane that covers the entire internal surface of the heart; It is made up of three different tunics, the innermost one is a true endothelium. The endocardium, due to its origin, corresponds to the vascular wall and its three tunics are related to the three vascular tunics: the external one, to the adventitia; the media, with the media and the internal with the intimate. The valves also have an endothelium that is supported by connective tissue. The tunics of the heart valves normally do not have blood vessels.

BIG VESSELS

The great vessels - aorta and pulmonary artery - have their own characteristics. The pulmonary artery has an approximate size of 50 mm. Almost immediately originates, it divides into two large trunks: the right and the left, the latter constitutes the extension of this artery.

The aorta is the largest artery in the body. First it goes upwards, ascending aorta; then backwards riding on the left bronchus, arch of the aorta; and finally, downward along the spine, the descending aorta, which is called the thoracic aorta, above the diaphragm, and the abdominal aorta, below it.

The coronary arteries are branches of the ascending aorta, whose orifices or ostia are found above the two anterior sigmoid arteries of the aortic orifice.

The arch of the aorta also gives rise to a series of branches: the brachiocephalic trunk, which divides a short distance into the right subclavian and right carotid arteries; The left subclavian and the left carotid arise directly from the arch.

Above the aortic valves there is a dilation: the aortic bulb, which contains three sinuses of Valsalva or aortic sinuses, also called the two anterior coronary sinuses, right and left. Between the arch and the descending aorta there is a narrowing called the aortic isthmus, often followed by dilation. A type of tubular area of fibrous tissue (annulus fibrosus) joins the left ventricle to the ascending aorta.

Other points of some value to consider with respect to arterial trunks are:

1. The division of the primitive carotid into internal and external. At the point where this division or bifurcation occurs, there is a slight dilation that constitutes the carotid sinus, which is located at the level of the neck, above the cricoid cartilage.
2. The division of the abdominal aorta into two large vessels - the common iliac vessels - occurs approximately 10 mm above the umbilical scar.

Structure of the arteries

Every artery has three layers:

1. The inner layer or tunica intima.
2. The middle layer or tunic media.
3. The outer layer or outer tunic.

The marked differences that exist in the structure of the tunica media facilitate the division of these vessels into two large groups: large arteries and small arteries.

Large arteries have the tunica intima made up of an endothelium that rests on connective tissue and contains different sheets. The tunica media is made up of a large amount of elastic tissue with a few cells, supported by ordinary connective tissue.

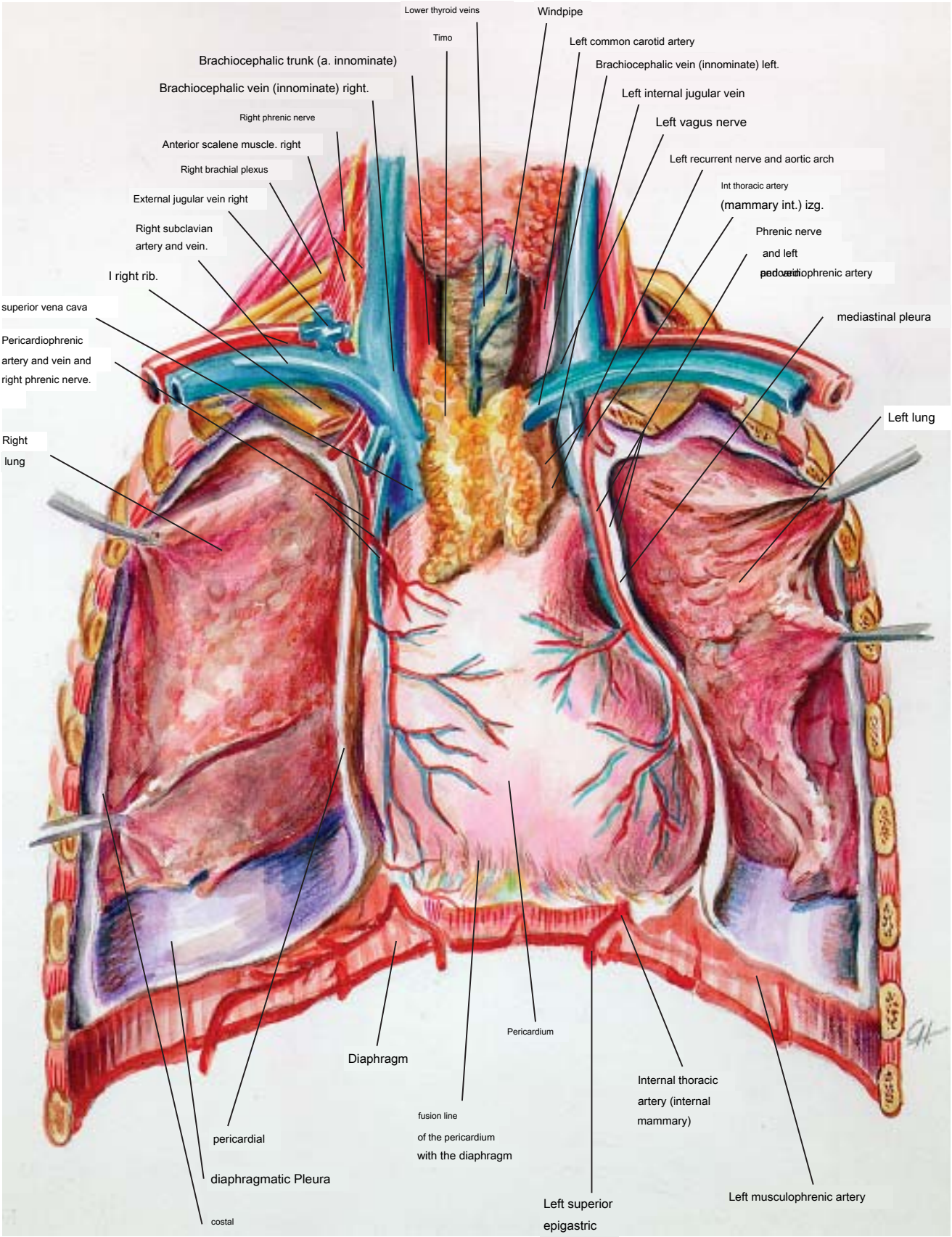


Fig. 10.2 Pericardium.

The small arteries have the tunica intima made up of an endothelium that rests on elastic tissue.
The tunica media is thick, typically muscular and covered by smooth cells that are arranged in a circular shape.
The external tunica or adventitia is thick and is formed by connective tissue that usually contains some elastic sheets.

The medium and large arteries have their own vessels in the external tunic; They can penetrate the tunica media and are called vasa vasorum.

The connection between the muscular tunica media and the elastic tunica externa of the arteries acquires, due to its function, special importance.

The nerves that innervate the small arteries form plexuses in the external tunic, between it and the media, and inside the latter.

The capillary wall is made up of endothelial cells joined by their edges and supported by a delicate reticular network of connective tissue with some Rouget cells, which should not be confused with muscular elements.

Structure of the veins

The venous walls contain three different kinds of tunics: the intima, similar to that of the arteries, has an endothelium with an elastic membrane; The media, which is much thinner than that of arteries of similar diameter, contains elastic and muscular elements in a network of loose or soft connective tissue; the external one, which is much thicker than that of the arteries and may contain muscular elements.

Veins may have membranous bags called valves, which are generally seen in medium-sized veins and veins in the extremities. These valves are made up of a double endothelium with a thin layer of connective tissue between its leaves.

Both large and medium veins have their own vessels that run through the external tunica and are also called vasa vasorum.

ARTERIES OF THE HEART

Two arterial vessels supply blood to the entire heart. They are the left and right coronary arteries, branches of the ascending aorta (fig. 10.3).

The left coronary artery partially surrounds the pulmonary artery, enters the interventricular sulcus from its anterior part and is prolonged by the descending artery anterior to the interventricular branch that runs towards the apex or vertex of the heart. This artery supplies blood to the anterior surface of the ventricles. A branch of the left coronary artery, the circumflex branch, shortly after its origin leaves the main trunk and rotates.

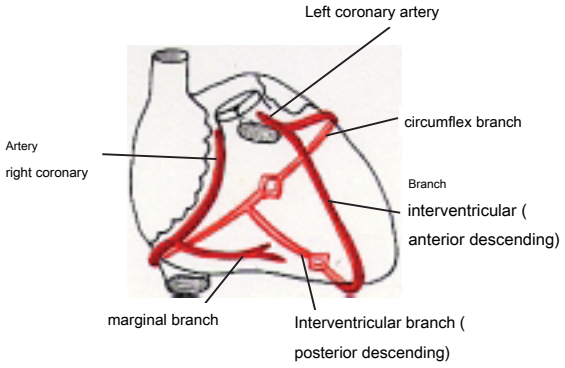


Fig. 10.3 Coronary arteries.

dea to the heart passing along the left atrioventricular groove.

The right coronary artery originates from the aorta and, following the anterior part of the right atrioventricular sulcus, reaches the posterior interventricular sulcus, where the posterior descending branch continues towards the apex. This artery supplies blood to the entire posterior surface of the ventricles.

In 80% of cases, each atrium is supplied by its respective coronary artery. On the contrary, the interventricular septum and the adjacent part of the ventricles always have a mixed irrigation: the right coronary artery supplies the posterior third of the interventricular septum and the left coronary artery supplies the anterior two-thirds of the septum.

The left artery supplies mostly the anterior part, while the right artery does the same with the posterior part.

The right and left coronary arteries originate in the aortic bulb, below the upper edges of the semilunar valves, therefore during systole, the entrance to the coronary arteries is closed by the valves and the arteries themselves are compressed by the contraction of the cardiac muscle, as a consequence the irrigation of the heart decreases during systole; Blood enters the coronary arteries during diastole, when the entrance orifices of these vessels are not closed by the semilunar valves.

The coronary network can be schematically divided into two sections:

1. The large trunks that run along the surface of the heart.

2. The small vessels that penetrate deeply into the myocardium and are subject to the effects of cardiac contractions.

There are a few anastomotic branches between the small coronary arteries that do not develop

exaggeratedly, except in elderly patients or when coronary heart disease occurs.

These communications allow blood to be transported in one direction and another and connect the branches of the right coronary artery with the branches of the left coronary artery. There is often a wide anastomosis between the capillaries of the two systems.

The distribution of the coronary arteries is not the same in all hearts; Different variants of the development of the coronary arteries are observed, which give rise to three forms of cardiac irrigation: a uniform one, with equal development of the two coronary arteries; another in which the right coronary group predominates, and the third in which the left coronary circulation predominates.

The S-A node receives blood supply from only one of the auricular arteries; Naturally, this can be variable depending on whether it is a branch of the circumflex right or left. This artery gives rise to a vascular ring that surrounds the orifice of the superior vena cava.

1st.

In addition to the conventional type of blood supply, the myocardium may be irrigated:

1. By extracardiac anastomoses.

2. By the Thebesius vessels.

3. Through an inversion of the circulation of the coronary veins.

HEART VEINS

Venous blood from the heart can circulate through different routes:

1. Through the great cardiac vein that ends in the coronary sinus, venous dilation that opens into the right atrium (fig. 10.4).

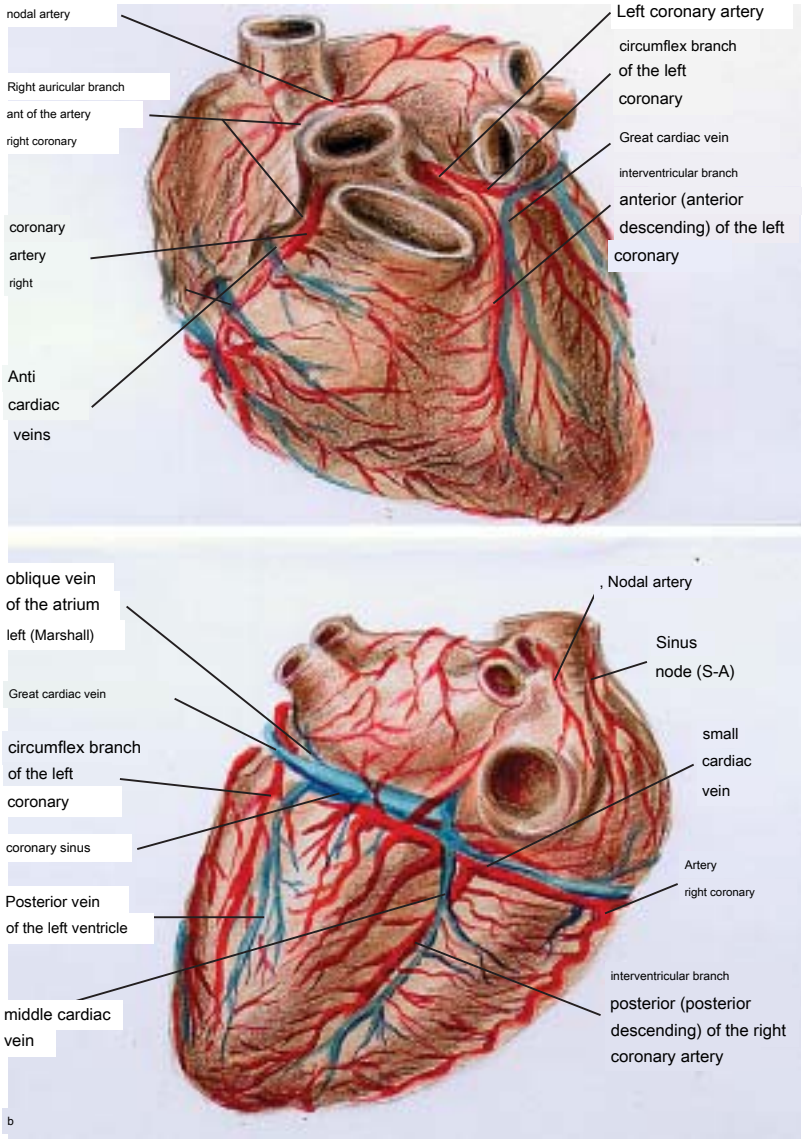


Fig. 10.4 Veins of the heart: a, sternocostal aspect; b, diaphragmatic face.

2. Through small veins that run along the surface of the heart and open independently into the right atrium.

3. Through the Thebesian vessels, which open directly into any of the four cardiac chambers.
4. By the luminar and arteriosinusoidal connections, which occur within the cardiac chambers.

LYMPHATIC VESSELS

The lymphatic vessels of the heart form an extensive subendocardial network that is more developed at the level of the ventricles. The largest collector of these vessels runs superficially through the grooves of the heart and connects with the lymph nodes located at the bifurcation of the trachea, the left collector; the right reaches the left anterosuperior lymph nodes of the mediastinum, located in the arch of the aorta.

Existing connections between the supradiaphragmatic and infradiaphragmatic networks may have some importance in the spread of bacteria from the peritoneum to the pericardium, or vice versa.

SENSORY NERVES OF THE HEART

Usual stimuli applied to the myocardium do not cause pain. However, lesions of the coronary system are a frequent cause of pain.

The innervation of the cardiac muscle, with a specific structure and function, is characterized by its complexity, since it is made up of multiple plexuses. The entire cardiac nervous system consists of the following parts:

1. Afferent and efferent trunks.
2. Plexuses in one's own heart.
3. Ganglion formations related to the plexuses.

The nerves of the heart can be functionally classified into four groups: inhibitors, accelerators, depressors and intensifiers. These groups of nerves make up the vagus nerve and the sympathetic system (Fig. 10.5).

The sympathetic nerves, mainly the postganglionic fibers, arise from the three cervical ganglia and the five upper thoracic ganglia. The superior cervical cardiac nerve, from the superior cervical ganglion;
the middle cervical cardiac, from the middle cervical ganglion; and the lower cervical cardiac, from the cervicothoracic ganglion or stellate ganglion.

The thoracic cardiac nerves arise from the thoracic ganglia of the sympathetic chain.

The cardiac branches of the vagus nerve begin in its cervical portion: superior cardiac branches; in its thoracic portion: the middle cardiac branches; and in the recurrent laryngeal nerve (branch of the vagus): the lower cardiac branches. These nerve branches when they reach the heart meet in two groups, the superficial and the deep, in intimate contact with the lymph nodes of the tracheal region, forming two plexuses, the superficial cardiac and the deep cardiac, which continue in the coronary plexuses and in the subepicardial plexus, from the latter the intraorganic branches.

The afferent nerve fibers begin in the receptors of the myocardium, pericardium, endocardium and in the coronary arteries and aorta, and, together with the efferent fibers, they extend, thus entering the structure of the vagus nerve and the sympathetic system.

There are also white rami communicantes that communicate the thoracic sympathetic ganglia with the posterior roots of the spinal cord.

CLINICAL PHYSIOLOGY

The heart is the organ that propels blood through the circulatory system. Each of the parts it consists of plays a special role in the production and use of its contraction.

In its mechanical operation there are two impeller pumps in parallel, one for the systemic circulation, the left, and another for the pulmonary circulation, the right.

MYOCARDIUM

The cardiac muscle has four fundamental properties: automatism or chronotropism, conductivity or dromotropism, excitability or bathmotropism and contractility or inotropism. These properties depend on its aerobic, and emerging anaerobic, metabolism, with the consequent transfer of ions through the myocardial fiber, during its states of action, recovery and rest. The various concentrations of ions occur on both sides of the membrane of the myocardial fiber, with the consequent differences in electrical potential, known as transmembrane potential, which has three successive phases: the rest potential, the action potential and the recovery potential, the first corresponding to diastole and the other two to systole.

Automatism or chronotropism

It is the property of producing rhythmic stimuli. Although present throughout the myocardium, this property is highly developed in the specific system, that is, in the pacemaker (which is the sinoatrial node) and in the conductive system, especially in its higher parts, as demonstrated in the different automatic rhythms.

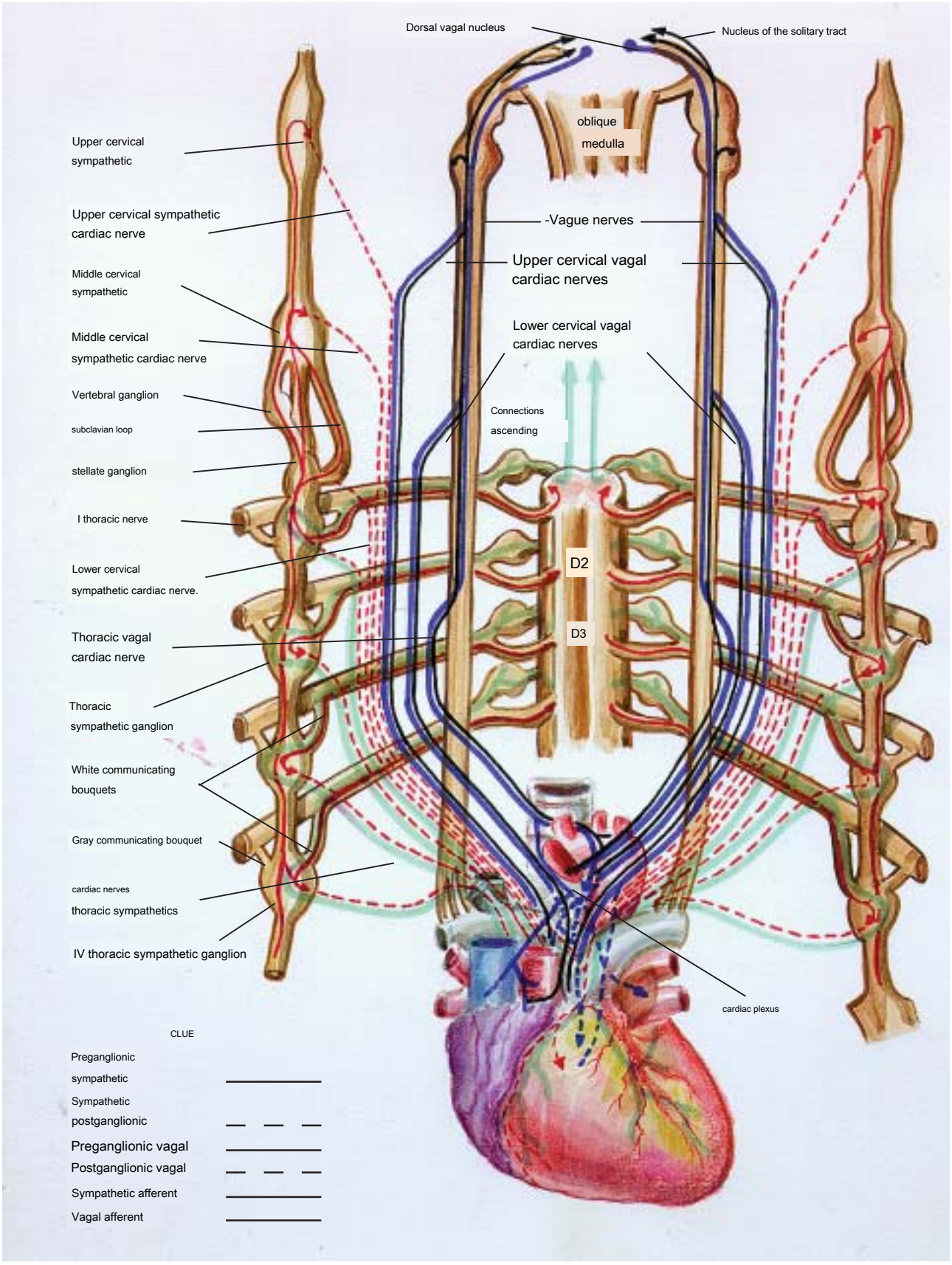


Fig. 10.5 Nerves of the heart.

Conductivity or dromotropism

It is the ability to receive and transmit stimuli. It is influenced by the length of the refractory period.

Excitability or bhathmotropism

It is the ability to react to certain stimuli. This property is common to all striated muscles, but has special characteristics in the cardiac muscle due to the long duration of the cardiac periods.

Fractalists.

Contractility or inotropism

It is the property of the muscle to shorten, which is common to all muscles: smooth and striated.

There is a difference between the automatism of the ordinary myocardium and that of the specialized myocardium, with the former maintaining the polarity of the negative resting potential inside the fiber in relation to the outside at a value of around – 90 mV, to remain more or less the same until new activation; while in the specialized myocardium (automatic or specific system of the heart) it gradually decreases. and when it reaches 60 mV, the threshold of automatism, self-excitation occurs, earlier in the sinus node than in the atrioventricular or idioventricular node: the former governs the cardiac rhythm, which is why it is called an effective pacemaker; the others that remain emergent, that is, the potential pacemakers, are then abolished.

The automatism of the sinus node ranges between 40 and 150 impulses per minute, that of the atrioventricular node is only 30-50 impulses, and that of the idioventricular node is 18-30 impulses per minute.

The already mentioned rest potential is followed by the action potential, which corresponds to the activation phenomenon, which begins with systole: a sudden total loss of the interior negativity of the fiber in relation to the exterior occurs, that is, a complete depolarization of the fiber. Next, it becomes polarized again, the recovery potential, and reaches the resting potential again, being totally inexcitable up to -60 mV, the limit between the absolute and relative refractory period.

The stimulus that originates in the sinus node spreads by continuity eccentrically, first to the walls of the right atrium, and then to those of the left.

After the atria are activated, the same stimulus reaches the atrioventricular node and discharges its automatism, initiating a new stimulus that is conducted by the bundle of His and activates the ventricular myocardium.

Laws on the functional properties of the myocardium Law of all or nothing

When the heart is stimulated, it reacts with maximum intensity, whatever the intensity of the stimulus. The same law is true for an isolated fiber of any skeletal muscle according to its different fibrous sheets. Strong stimuli force a greater number of fibers to contract and also cause a much greater contracture. Due to the syncytial structure of the myocardium, all normal cardiac fibers have the same reaction and, therefore, contract as a unit.

Variations in the excitability and contractility of the cardiac muscle due to mechanical stimuli (initial size of the fibers), chemical stimuli (concentration of H ions), or physiological phenomena (changes in blood supply, fatigue), can cause different intensity responses to the stimulus. This explains the apparent exceptions to this law, which becomes true when the condition remains constant.

Law of refractory period

The extreme shortness of the period during which the skeletal muscle is not excitable (refractory period) would cause a gradual reaction of contractions and the appearance of tetanus. This is impossible in normal cardiac muscle. During contraction, the cardiac muscle does not react to abnormal stimuli (absolute refractory period). After the end of the contraction the cardiac muscle becomes more or less excitable (relative refractory period), until the excitability ends up being entirely normal.

Law of influence of initial size

Distension of the chambers of the heart due to an increase in blood flow increases the length of the cardiac fiber and tends to cause proportionally greater contractions. This property is also recognized as Starling's Law and is common to all striated muscles.

PHYSIOLOGICAL NEEDS OF THE HEART

The mammalian heart, when isolated and perfused, maintains continuous and rhythmic contractions under certain conditions. The infusion liquid must contain oxygen, be maintained at a temperature similar to that of the body and have a defined pressure.

The oxygen consumption per hour of the heart of the isolated mammal is approximately 3.24 mL/g of the organ under conditions similar to those existing in the organism at rest.

Temperatures between 26 and 40 °C are well tolerated and cause proportional variations in heart rate.

The isolated heart is very sensitive to changes in the reaction of the medium (pH), because it only has a limited buffer system, so that

A small concentration of lactic acid of 0.7% causes severe arrhythmia.

The composition of the fluid is important to keep the heart alive. Calcium in excess or in a normal concentration, but in the absence of potassium, increases the duration of systole and, finally, stops the heart in this phase (calcium rigor).

Potassium in excess or in normal concentration, in the absence of calcium, increases the duration of diastole and subsequently stops the heart in this phase (potassium inhibition). An increase in alkalinity (high pH) acts the same as an excess of calcium: and a decrease in alkalinity (low pH) acts the same as an excess of potassium.

As the concentration of sodium is essential in the perfusion liquid, it is necessary that there be a perfect relationship between calcium, potassium and sodium to keep the myocardium functioning properly.

Carbon dioxide has a very complex action.

Apart from its action on the spinal centers (stimulus on the respiratory, vasomotor and vagal centre), an excess of carbon dioxide acts on the myocardium, depressing automatism and conductivity; on the contrary, an insufficient concentration tends to shorten diastole, make it incomplete and reduce cardiac output.

Cardiac muscle metabolism

The heart metabolizes glucose, fat, and perhaps amino acids and lactic acid.

Glucose serves partly to replace spent glycogen, and partly is consumed.

Lactic acid is absorbed from the blood of the coronary arteries, but its use has not yet been demonstrated. Fats replace carbohydrates when they are scarce, and it has not been proven that amino acids, as such, are used by the heart.

The process of myocardial contraction is due to the interaction that exists between a protein complex, ACTOMYOSIN (actin plus myosin), and a water-soluble nucleotide, which is adenosine triphosphate (ATP). Myosin binds to ATP through magnesium and potassium ions. When there are high concentrations of salts in the myocardium, the myosin-ATP complex remains separated from the actin protein fraction; On the contrary, when concentrations are low, actin and myosin-ATP bind. This relationship in the ionic concentration necessary to provoke this reaction basically depends on the chemical changes caused in the cardiac fiber by the electrical depolarization wave that travels through the myocardium in each cycle. As long as actin and myosin-ATP are kept separated, the myocardial fibers remain elongated and have a high energy power (dilation). On the contrary, when these factors

When they join together, dehydration and shortening of the fibers with low energy power (contraction) occur.

If in these circumstances any process prevents the shortening of the fiber, a large increase in tension is caused.

The chemical reactions that take place in the myocardium are similar to those that occur in any skeletal muscle. They can be summarized as follows:

a) Adenosine triphosphate decomposes into adenylic acid and phosphoric acid (this reaction does not require oxygen) .
b) Phosphocreatine is split into phosphoric acid plus creatine (does not require oxygen consumption).
c) Glycogen is converted into lactic acid (does not require oxygen); Part of the energy resulting from this reaction is used for the resynthesis of phosphocreatine.
d) Lactic acid is consumed leading to the formation of carbon dioxide and water (oxygen is necessary for this reaction); Part of the resulting energy is used for glycogen resynthesis.

The conversion of glycogen to lactic acid requires a large number of enzymatic reactions. Under physiological conditions, muscle glycogen is split into lactic acid and water, producing a large amount of energy. Among the different steps, one of the most important is the one that tends to form pyruvic acid.

When there is a relative decrease in oxygen, lactic acid increases.

In the animal, both glycogen and lactic acid can be carried to the heart by the blood, given that their synthesis can take place in other places in the body. An important difference between cardiac muscle and skeletal muscle is that the former cannot tolerate a large oxygen deficit. Therefore, cardiac contraction does not occur if the heart does not receive an adequate and continuous flow of oxygen.

Heart pacemaker and conduction system

In man, the pacemaker is located within the sinoatrial node (S-A node) or in neighboring tissue.

Apparently the activity of the pacemaker, which is the cause of the heartbeat, is the result of a spontaneous development of rhythmic oscillations of electrical potential.

When a potential change of sufficient intensity occurs, an electrical discharge begins and propagates, which causes stimulation of the neighboring myocardium. The conduction of this discharge follows the myocardial fibers at different speeds, which are directly proportional to the size of the fiber.

The stimuli produced by the activity of the pacemaker usually reach the most distant points

of the heart before any other stimulus can develop.

The impulse propagates as a wave in the wall of the right atrium, with a speed of 600-1200 mm/s.

so the right atrial appendage receives it at approximately 0. 03 s. Since the left atrium is excited only 0.045 s after the initiation of the impulse, there is a small time difference between the excitation of the right and left atrium. This demonstrates the great speed of interatrial transmission.

For years there has been discussion about the existence of a special conduction system between the sinus node and the atrioventricular node. At present, three fascicles or fine tracts have been identified that make this connection (fig. 10.6):

1. Anterior internodal tract.

2. Middle internodal tract.

4. Posterior internodal tract.

Some of these tracts are further subdivided.

The propagation wave undergoes a well-defined arrest at the level of the atrioventricular node; To date, it is unknown whether this is caused by a specific resistance at its level, or because said impulse is sent to another still unknown center. From this point the electrical wave progresses at great speed.

speed through the bundle of His and its branches. As a result of the characteristics of the septum, and especially its endocardial surface, the septum is excited first and then the papillary muscles, and approximately 0.04 s after the stimulus begins at the starting point of the atrioventricular node, the entire endocardial surface of the ventricle has been activated, with the right ventricle slightly ahead of the left.

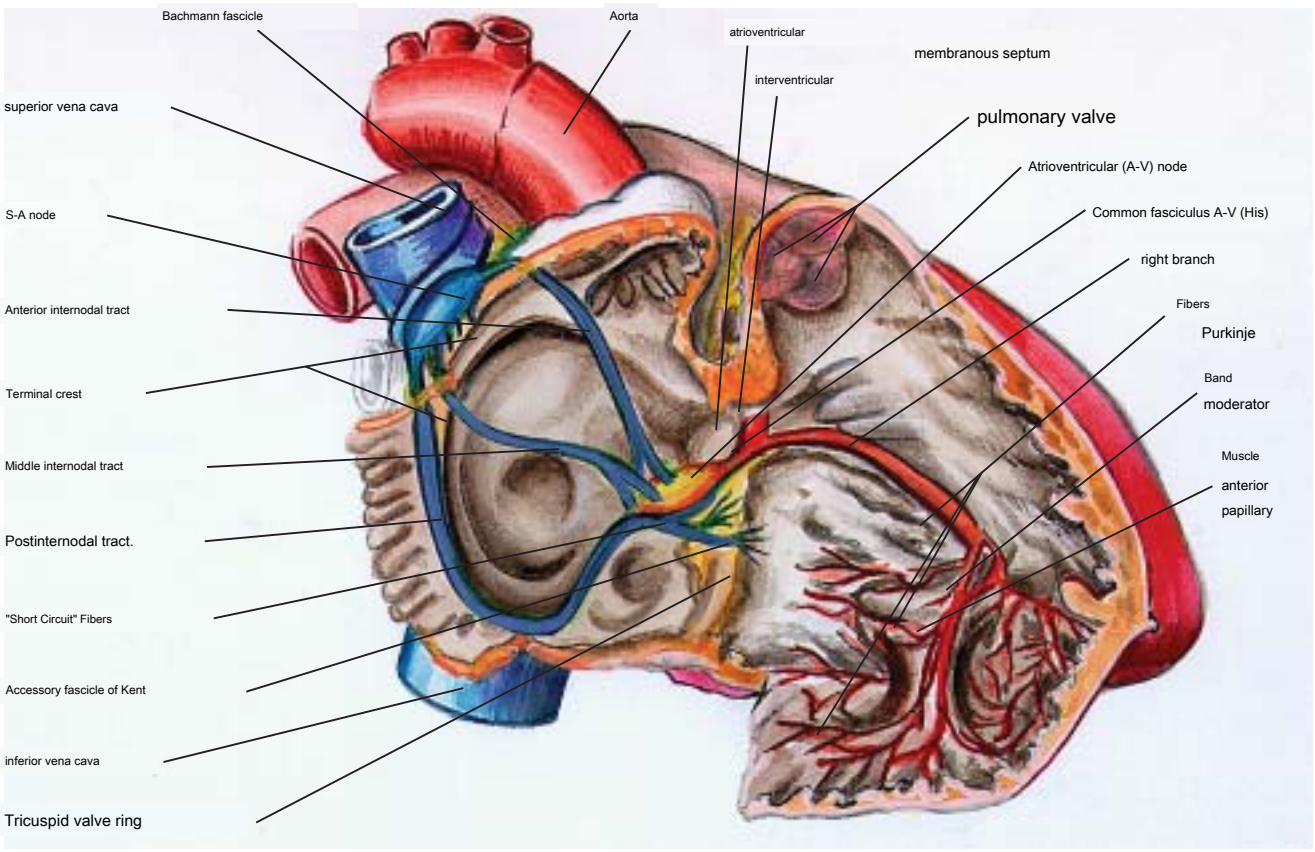
The upper part of the interventricular septum is stimulated first, then the apex or tip of the ventricles, and then their base.

The transmission of the impulse from the endocardial surface to the rest of the myocardium follows the fibers of the Purkinje system, which penetrate almost to the epicardium.

When the downward propagation wave is interrupted and does not reach the different parts of the myocardium, a lower center becomes the heart's pacemaker. Experimental and clinical evidence reveals the automation of the different centers, the frequency with which they are capable of originating the stimulus and the different speeds that each of them develops:

S-A node.....	40-150 impulses per minute
A-V node	30-50 impulses per minute
Fascicle of His....	18-30 impulses per minute

Fig. 10.6 Internodal tracts: right side.



The study of the electrocardiogram has extraordinarily increased knowledge of the excitation processes of the heart.

Mechanism of cardiac action

The phenomenon of cardiac contraction and dilation has been studied in different ways:

a) Introducing manometric tubes into the cardiac cavities and measuring the different pressure changes that take place in them.
b) Investigating the activity of the different parts of the heart in the open chest experimentally, both through the direct transmission of movements and through volumetric changes.
c) Observing the movements of the chest wall caused by the activity of the heart.
d) Analyzing the movements of blood flow and reflux of the heart and those of the respiratory system due to the effect of cardiac action.

The cardiac cycle begins with the contraction of the atria and is called atrial systole. The excitation is followed by a contraction wave in a downward direction that produces efficient propulsion of blood volume towards the ventricles.

Venous reflux is prevented by muscle contraction at the opening of these vessels, and also by venous pressure, which in this phase is slightly higher than in the atrium.

Atrial contraction is not very marked and is rather revealed during the opening phase of the atrioventricular valves by the movement of blood flow from one chamber to the other.

Atrial contraction is not essential because most of the ventricular filling occurs at the beginning of diastole. However, it complements 1ventricular filling and contributes to the normal function of the atrioventricular valves. In cases of tachycardia and mitral stenosis, atrial contraction may acquire greater importance.

The initiation of ventricular contraction increases the pressure in these cavities and in the atrioventricular valves (tricuspid in the right heart and mitral in the left). Immediately afterwards, the contraction of the papillary muscles prevents the inversion of the valves and allows a greater increase in pressure until it exceeds that existing in the aorta and pulmonary artery. In this short period, ventricular contraction causes a rise in pressure without blood being mobilized. This extraordinarily brief phase is called the tension period or isometric contraction period, because the muscle fibers of the ventricle come into tension without shortening.

As soon as the ventricular pressure exceeds that in the arteries, the semilunar valves open and blood flow begins (Fig. 10.7).

During this period of flow or blood injection, the fibrous septum that supports the atrioventricular valves shortens due to the contraction of the ventricles and exaggeratedly increases the size of the corresponding atrium, causing aspiration of blood from the veins. From beginning to end, the ventricle maintains a constant pressure, which is manifested by a type of flattened line in the intraventricular pressure tracing.

Despite their apparently delicate structure, the leaflets of the atrioventricular valves have great resistance and strength. When they are closed, they not only come into contact, but form an intimately united surface without the slightest folding. The closure that is initiated by blood reflux is increased by the ventricular contraction that immediately occurs. Inversion of the valves is prevented by the action of the chordae tendineae aided by the papillary muscles. The fact that the septal muscles are the first to contract together with the papillary muscles ensures timely closure of the valves.

The termination of atrial contraction contributes to the closure of the atrioventricular valves, since the reflux of blood flow and the reversal of the pressure gradient force the valves to adopt their position. This fact is demonstrated in cases of incomplete atrioventricular block with the appearance of the symptoms of periodic and temporary insufficiency that occur.

In cases of delayed atrioventricular conduction there may be a double closure of the atrioventricular valves; the first at the end of atrial contraction and the second at the beginning of ventricular systole.

The semilunar valves of the aorta and pulmonary artery resemble small bags attached to the walls of the vessels. The blood contained in them keeps the valves away from the wall.

Both the reversal of the pressure gradient created by the sudden cessation of the blood column and the reflux of the current determine the closure of these valves at the end of ventricular systole.

Each ventricle can be compared to a pump that maintains the circulation of blood flow in only one direction, due to the existence of valves.

During ventricular contraction all diameters of the heart decrease, the base is directed downward, the great vessels narrow, but the apex does not move upward. The spiral arrangement of the muscular bands of the ventricles causes their contraction

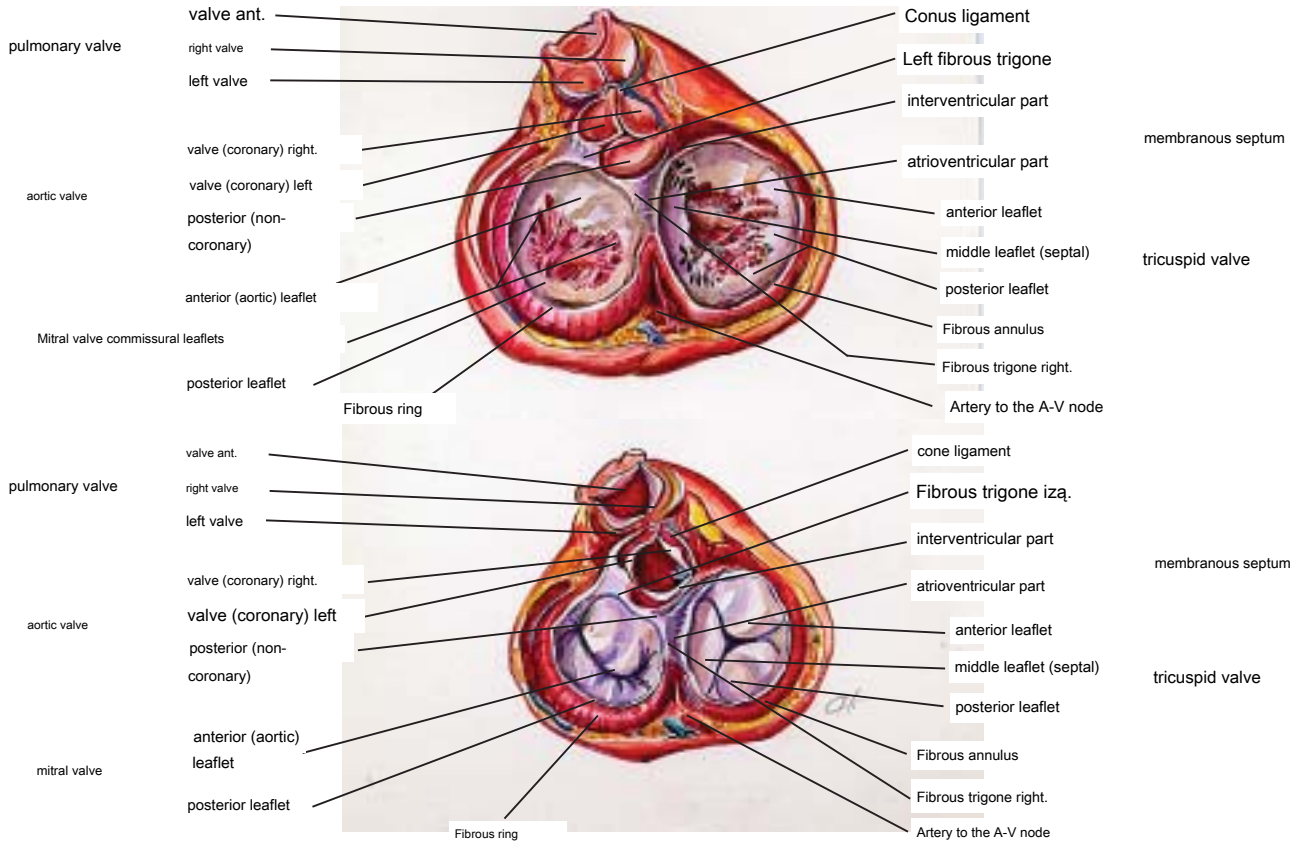


Fig. 10.7 Position of the heart valves with the atria removed: a, the heart in diastoles, seen from the base; b, the heart in systole, seen from the base.

be very efficient and also force the heart to rotate to the right, resting its apex more firmly against the chest wall; This, together with the increased tension of the ventricular mass, causes the apex beat.

At the end of ventricular systole the pressure drops to zero. Subsequently, and after a short period of latency (the so-called protodiastole), the semilunar valves of the aorta and pulmonary artery close. A short time interval separates this phase from the subsequent opening of the atrioventricular valves and is called the isometric relaxation period.

Ventricular filling begins after this period, that is, following the opening of the atrioventricular valves.

The interventricular septum has an important role in the dynamics of this movement and a large part of the right ventricular contraction is probably due to this structure.

The filling of the ventricles has the following characters:

tes:

a) An initial phase of rapid filling (early diastole), which is caused by the pressure difference between a distended and full atrium and an empty ventricle.

Studies aimed at demonstrating the existence of active ventricular diastole in this phase have been inconclusive. In this phase, the entire ventricular reservoir experiences a drop in tension due to the accelerated passage of blood flow through the opening of the atrioventricular valves.

b) A slow filling phase (diastole). The gradual filling of the ventricles decreases blood flow and there is a gradual increase in pressure in the venoatrial reservoir.

c) A final phase of rapid filling (presystole) produced by atrial contraction that complements ventricular filling. The veins and atria show a sudden increase in pressure and as soon as the atrial contraction is complete, the ventricles begin to contract because the stimulus has already reached the myocardial muscle at this level.

The following time intervals in seconds can be considered typical of a heart with a speed of 68 beats per minute:

ventricular systole	
Strain	0.06
Maximum expulsion	0.12
Minimum expulsion	0.16
Total sum	0.34

Ventricular diastole	
Protodiastole	0.04
Isometric Relaxation Full	0.07
Fast	0.10
Slow full	0.20
Atrial dynamics	
Dynamic range	0.05
Atrial contraction ..... 0.06 Total sum .. 0.52	

In summary, we can point out that the cardiac revolution consists of the successive periods or phases that we will describe below.

Ventricular systole:

1. Isometric contraction. It corresponds to the contraction without shortening of the muscle fibers of the ventricle, beginning at the moment the atrioventricular valves (tricuspid and mitral) close. During this phase the ventricle forms a completely closed cavity; The atrioventricular valves are already closed and the sigmoid valves (pulmonary and aortic) have not yet opened. The isometric contraction ends when the intraventricular pressure, which has risen very quickly inside, reaches the pulmonary and aortic pressure, causing the sigmoids to open.

2. Ventricular ejection. It is fast at first and then slow. It begins with the opening of the sigmoids. During this phase the ventricles contract and expel blood towards the pulmonary artery and aorta. When the sigmoids are open, the ventricles with their corresponding arteries constitute a common cavity whose internal pressure is the same. This phase ends when ventricular pressure drops enough for the sigmoids to close.

Ventricular diastole:

1. Isometric relaxation. It begins with the closure of the sigmoids. During this phase the ventricles once again constitute closed cavities as the atrioventricular valves are still occluded. Intraventricular pressure drops rapidly. This phase ends when the pressure inside the atria exceeds that of the ventricles and causes the atrioventricular valves to open.

2. Ventricular repletion or filling It begins with the opening of the atrioventricular valves. It can be subdivided into three parts:

   a) Rapid ventricular repletion or filling: occurs at the moment the atrioventricular valves open and corresponds to the sudden inrush of atrial blood into the ventricles.

   b) Diastasis: corresponds to an increasingly slower ventricular repletion or filling.

   c) End-diastolic period: characterized by atrial contraction that completes the filling of the ventricles and is manifested on the ECG by the appearance of the P wave. During this period the ventricle remains in diastole. The end of ventricular diastole occurs at the moment when the pressure in these cavities, rising suddenly, causes the closure of the atrioventricular valve apparatus.

HEART NOISES

Auscultation of the normal heart reveals two sounds, occasionally three. Translation of heart sounds by phonocardiogram may reveal fourth and even fifth sounds (Fig. 10.8).

The first sound occurs at the beginning of ventricular systole and corresponds to the tension period and the beginning of the expulsion period.

The second sound is much shorter, begins at the end of systole and corresponds to the isometric relaxation period; The name systolic sounds has been suggested for these two constant heart sounds.

The other two sounds that can be heard less frequently occur during diastole. The name diastolic sounds has been suggested for them.

Systolic sounds

First noise. Initiation of ventricular systole, closure of the atrioventricular valves and opening of the semilunar valves.

Second noise. End of ventricular systole, closure of the semilunar valves and opening of the atrioventricular valves.

Diastolic sounds

Third noise. Rapid filling of the ventricles.

Fourth noise. Active and rapid filling of the ventricles, due to atrial contraction.

Let us remember that the continent and the content participate in the origin of heart sounds, because their components are supportive.

Complex corresponding to the first noise

It has been proven that the two main components of the first sound coincide with the ventricular waves (QRS) of the electrocardiogram and with the tracing corresponding to intraventricular pressure; All of these elements manifest two valve events: the closure of the atrioventricular valves and the opening of the semilunar valves.

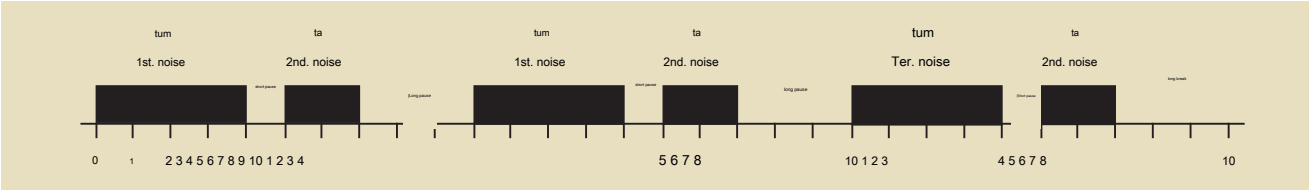


Fig. 10.8 Heart sounds.

It has also been shown that the muscular vibration of the empty heart is very mild and difficult to hear; Despite this, the first noise is the result of these factors: muscular and valvular. Sudden changes in muscle tension first activate the atrioventricular valves and then the semilunar valves.

This rapid succession of events causes a double vibration of the heart muscle that is transmitted to the chest wall.
In other words, the first audible noise is the expression of the distinct movements of the heart, which are reflected during the first part of the tip shock.

Complex corresponding to the second noise

It is mainly caused by the closure of the semilunar valves and the transmission of vibrations to the heart and chest wall; Furthermore, these vibrations of vascular origin, as well as the opening of the atrioventricular valves, contribute to their formation in certain cases.

Complex corresponding to the third noise

The third sounds originates in the ventricular wall, as a result of the vibration caused by the passage of blood at the time of rapid passive filling of the ventricle.
The third sound has been attributed to valve vibrations, a theory that at present is only of historical interest, or to the apical impact on the chest wall; This, of course, can only be a concurrent factor.

A fourth and fifth noise have also been described, but they are of no real importance clinically.

RESIDUAL VOLUME AND CARDIAC OUTPUT

The large thoracic and abdominal veins represent, together with the right atrium, a venous reservoir that contains a large amount of blood at low pressure.

The amount of blood pushed into the aorta in each ventricular contraction is called contraction volume or systolic discharge, and varies between 70 and 80 mL of blood. A similar amount is removed from the systemic venous reservoir with only 3-5 mm of variation in right atrial pressure.

The mean volume of the right ventricle is probably about 50 mL of blood at the end of systole, and about 130 mL at the end of diastole; This means that in most cases about 50 mL of blood residual remains within the right ventricle under normal conditions.

The pulmonary veins and the left atrium represent a pulmonary venous reservoir that is made up of the blood contained in the lungs. This reservoir is much less compliant than the systemic one, and therefore, its pressure changes are broader.

The left ventricle contains approximately 50 mL of blood at the end of systole, and approximately 130 mL at the end of diastole; This means that about 50 mL of residual blood remains within the ventricle at the end of systole. The residual blood in the heart at the end of systole is approximately 260 mL; 100 mL seems to correspond to the ventricles, and the rest to the atria.

Multiplying the systolic discharges by the heart rate per minute gives the cardiac output or minute volume.

It is apparent that the heart can increase its output in one of three ways:

a) By increasing the systolic discharge, maintaining a constant speed or a constant frequency.
b) By acceleration of the rate per minute, maintaining a constant systolic discharge.
c) Due to reflex changes in ventricular contraction, which are independent of changes in the right atrium. This is possible due to the existence of residual blood within the heart and the pulmonary reservoir, and is frequently related to the effects of epinephrine.

Cardiac output or minute volume is a fundamental determination in our current knowledge of hemodynamics, it is based on these two formulas:

GC = VS FC Where: [1]

CO- cardiac output;
VS- stroke volume;
HR- heart rate.

TA = GC RP [2]

Where:

BP-blood pressure;
RP-peripheral resistance.

By solving the formula [1] we can find out the stroke volume of the left ventricle, and with the formula [2] we can find even the value of the peripheral resistance in a given patient.

For all of the above, the clinical search for cardiac output values guides the most up-to-date hemodynamic investigations.

Years ago, central venous pressure (CVP) values were considered basic for deciding the therapeutic approach to a patient in shock or heart failure. The Swan-Gang catheter has made it possible to measure pulmonary wedge pressures and three methods have been developed to directly obtain cardiac output at a given time, that is, the value of the heart's efficiency as a pump:

1. Fick's direct method. Based on the calculation that for every 100 mL of blood that passes through the lungs, they capture 4 mL of oxygen.
   This method is used in very specialized centers; It is arduously laborious and not always
   exact.
2. Indicator dilution method. It measures cardiac output using a dye that combines with plasma proteins and, therefore, does not pass from the blood to the tissues. A dose of the dye is injected into a peripheral vein, and seconds later, its concentration is detected and measured in a peripheral artery. Quantification is laborious, however it is widely used in Experimental Pharmacology through the use of electronic equipment that determines the concentration of the dye, for example, in the rabbit's ear.
3. Isotopic method. It is a very exact method. A dose of radioactive isotope is injected into a vein and then quantified in an artery; These values are taken to a calculator that, using a formula, determines the cardiac output in a few seconds. This method still lacks the simplicity that would make its use perfect at the bedside of seriously ill patients. The average cardiac output in men, under basal conditions, is 3.2 L/m² of body surface in one minute. This figure will be more exact if we relate it to body weight; It is approximately 76 mL/kg of weight. An average individual has a cardiac output of 5.6 min, with a frequency of approximately 66 beats per minute, and a systolic discharge of 84 mL.

Cardiac output is reduced when the subject is standing; It is increased by the ingestion of liquids and food. Physical exercise also increases pulse speed and systolic discharge, this being possible due to the great increase in venous return.

BLOOD VOLUME0. CIRCULATION TIME

The blood volume of mammals can be determined, approximately, on the basis of a percentage of body weight. In healthy individuals, plasma volume constitutes 5% of body weight when the hematocrit gives a value for venous blood between 40 and 50%.

Circulation time can be measured from a peripheral vein to a peripheral artery, or from a vein to the lung, or from the latter to an artery.

The average time from arm to tongue is 14 s, from arm to lung is 7 s, and from lung to cheek is 6 s.

Blood flow has been determined in several organs with the following result: kidneys, 1.3 L/min; brain, 0.8 L/min; extremities, 1.8 L'min; liver, 1.3-1.5 L/min.

HEART RATE

Heart speed varies with your size and weight.
This general law is true not only in human beings at different ages and sizes but also in animals of different species.

Heart rate decreases regularly with the increase in heart weight, following the growth of the organ from childhood to maturity. The duration of ventricular systole and the P-R interval increase at the same time.

The average pulse rate of the healthy individual is between 61 and 64 beats per minute, if taken under basal conditions.

The average can vary between 50-100 with a low speed for trained individuals, or with an elevation for those with decreased excitability of the vagus nerve and increased metabolic expenditure. Women have 7 or 8 more beats per minute than men.

PRESSURE IN THE CAVITIES OF THE HEART

The pressure in the right atrium is higher than the intrathoracic pressure, but very close to atmospheric pressure. It has been found that in a recumbent and resting individual, the pressure of the right atrium varies between 0 and more than 8 mm Hg. The left atrial pressure is slightly higher. The right ventricle has a systolic pressure of more than 20 to more than 25 mm Hg. The left ventricle has 110-150 mm Hg. In both ventricles the diastolic pressure is 0 mm Hg, or at most it reaches 5 mm Hg.

Deep inspiration markedly affects the pressure of the right heart chamber. Right atrial pressure can fall to less than 0 mm Hg, while right ventricular pressure varies between more than 20 and less than 6 mm Hg. On the contrary, cough

It produces an increase in right atrial pressure, up to 60 mm Hg, and in right ventricular pressure, up to more than 80 mm Hg.

The weakness and thinness of the walls of the right atrium and its compliance allow the filled volume of this cavity to be approximately twice that of the left atrium.

Because ventricular contraction lasts a small fraction of the total cycle (approximately one-tenth of a second), the atrium remains dilated during most of ventricular diastole and during ventricular systole, acting as a reservoir for blood reaching the heart. The traction caused by the ventricular muscle on the atrioventricular junction dilates the atrium and produces a kind of suction on the veins during ventricular systole.

The atrial appendage appears to have a very slight propulsive function, and serves as a complementary space that fills the niches at the base of the heart during ventricular systole.

In conditions other than normal, such as in the case of an accelerated rhythm, diastoles become extraordinarily short. In such cases, atrial contraction includes almost all of diastole and becomes more important.

NERVOUS AND CHEMICAL CONTROL OF CARDIAC ACTIVITY

The efferent nerve fibers that reach the heart depend on the vagus nerve and the sympathetic ganglia.
The vagal fibers end in a ganglion located in the wall of the atrium. From here the postganglionic fibers carry the impulses to the sinoatrial node and the atrioventricular node, the atrial muscle and the bundle of His. The right vagus emits a large number of fibers to the sinoatrial node; the left vagus, to the atrioventricular node.

Preganglionic fibers of the sympathetic system leave the thoracic section of the spinal cord and terminate in three cervical ganglia, and in the first five dorsal ganglia, however, only postganglionic fibers emerge from ganglia D2 through D5. These cause accelerating effects on the heart.

As the sympathetic fiber only reaches the atrial myocardium, ventricular function is not modified by the sympathetic fiber, but only through the conduction system.

The vagus nerve has a predominant and persistent action on the heart, as demonstrated by the acceleration of the heartbeat that follows the elimination of all autonomic nerve fibers. It decreases the heart rate by acting on the heart's pacemaker, producing a depression of automatism. Weakens atrial contraction, depresses contractility and shortens the refractory period of the atrium, increasing excitability. The amplitude and duration of the action current obtained from the auricular muscle is rapidly reduced by stimulation of the vagus.

The vagus nerve decreases the speed of transmission of impulses through the conduction system, depressing conductivity. The ventricles are indirectly affected by the increased extent of diastole, which increases the full phase and results in a longer interval, during which premature contractions can occur.
However, there is some evidence that the vagus exerts a direct influence on the ventricles.

The response to vagal stimulation varies with the rhythm existing in the atrium; If the rhythm is fast, normal or slow, the lazy person makes it even slower. On the contrary, if the atrium contracts very rapidly, 400 or more beats per second, vagal stimulation increases the discharge rate and causes atrial fibrillation.

The normal heart can temporarily stop due to excess vagal stimulation. However, this arrest is not permanent, since automatic ventricular contractions quickly begin to occur.

Stimulation of the right vagus can cause temporary arrest of the entire heart due to action on the pacemaker (cardiac arrest). Stimulation of the left vagus can cause temporary arrest of the ventricles by blocking the stimuli in the conduction system (action on the atrioventricular node and the bundle of His).

The action of the accelerators (sympathetic fibers) is less marked. Stimulation of these nerves causes an increase in heart rate (stimulation of automatism) and an increase in the contraction force of both atria and ventricles (stimulation of contractility). The efficiency of the cardiac muscle probably increases through a decrease in pH.

Both the action of the cardiac vagus nerve and that of the sympathetic nerve are produced by the release of chemical substances.

It is universally accepted that all autonomic and skeletal nerves act by the release of chemical substances.

The cardiac vagus acts by the release of acetylcholine, the sympathetic one by a substance similar to epinephrine or adrenaline. Acetylcholine, which acts by modifying the electrical behavior of the cell membrane, is quickly inactivated by a cholinesterase present in the blood, and therefore, its action is limited to the site of production.
On the contrary, sympathin diffuses through the bloodstream and is carried to all parts of the body, where it causes prolonged effects that are added to those of epinephrine.

11

EXPLORATION OF THE CIRCULATORY SYSTEM.

PRECORDIAL REGION

General guidelines

In this chapter we will consider the exploration or examination of the heart, clarifying that when we refer to the examination of the heart, we always include the examination of the large vessels that leave or reach it; That is, from the aorta and the pulmonary artery - arterial vessels that leave the left and right ventricle, respectively - and from the cava and pulmonary veins that reach the right and left atrium, respectively. As the heart and these great vessels are contained within the tőthorax, occupying the portion called the mediastinum, and within it the anteroinferior portion of the same, the physical examination of the heart and the great vessels will essentially include the anterior region of the thorax, which because it is located in front of them is called the precordial region.

The large vessels that arise from the heart, due to their branches or the course of their path - such as, for example, the aorta - contract relationships with regions close to the precordial region, such as the neck region and the epigastric region, above all; That is why the examination will include not only the precordial region, but also these regions.

Environmental conditions

The examination should take place in a quiet room, to better auscultate the heart sounds, and the necessary privacy and comfort should be obtained to avoid committing iatrogenesis and comply with the principles of medical ethics.

Adequate lighting is necessary, behind the observer. Tangential lighting from the side, such as from a gooseneck lamp, is effective in causing shadows on the anterior chest wall and, therefore, making the heartbeats in the chest more visible.

Exposure

We must leave the region we are going to explore uncovered, preferably from the navel upwards. If it is a woman, the breast must be partially uncovered, except during the inspection, when the entire area must be observed. It is not advisable to hear heart sounds through clothing.

Positions

The examiner should be placed to the right of the examinee, as this will be in a more favorable position to perform the basic examination methods, mainly palpation.

The examination must be performed with the person lying on the stretcher or in bed, which will allow them to change position, mainly to the left lateral decubitus and to the sitting and leaning forward position. A complete examination of the precordial region should include at least these three positions.

The examinee should adopt a comfortable position, lying down or better reclining. The supine position, raising the upper torso to a 30° angle, with the head resting on a pillow, the muscles relaxed, and with the arms resting comfortably at the sides, will be appropriate for most of the exam, so the exam should begin with the person in that position.

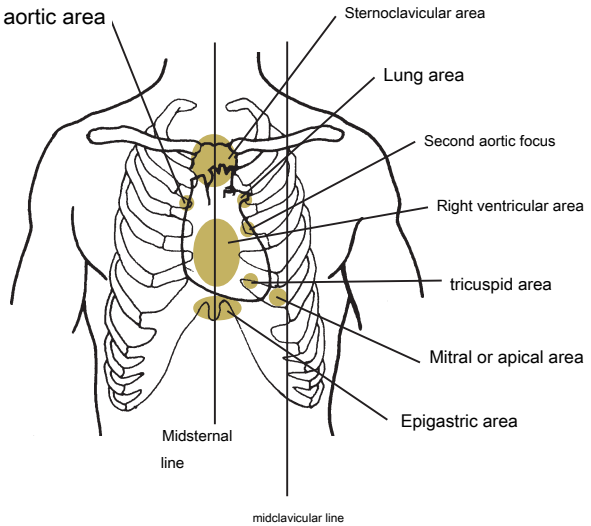
Changing the person's position during the exam is done to bring the underlying cardiac structures closer to the chest wall. The left lateral decubitus position will allow the tip to get closer to the chest wall and is best for detecting mitral valve murmurs. The forward sitting position will bring the base of the heart closer to the chest wall and is most effective for evaluating thrills and murmurs.

Precordial areas or foci

As the heart and great vessels are not visible, a system of precordial areas or foci is used (Fig. 11.1) to guide the examination and specify the location by describing each sound or pulsation detected during the examination.

exam.

Heart sounds are created by valve movements and blood flow in the heart. It is de- Fig. 11.1 Precordial areas and foci.



They detect the chest wall, but the focus where you feel or hear a sound may not be where it originates. This is because blood flow transmits sound beyond the point of its origin. Heart sounds originating from the valves are detected in the direction of blood flow in one of the following four main areas or foci in the wall:

Aortic focus. Located in the second right intercostal space, exactly next to the right edge of the sternum. It represents the direction of blood flow from the aortic valve and the direction of sound transmission that follows the closure of said valve.
Findings related to the aortic valve can be heard, although it is not the only thing heard, at this point.

Pulmonary focus: Located exactly next to the left edge of the sternum, in the second left intercostal space, it represents the pulmonary valve, which is located slightly lower than the second intercostal space. This limit correlates with the outflow tract of the pulmonary valve.

Tricuspid focus. Located in an area of approximately 3-4 cm, to the left of the sternum in its lower part, at the level of the fifth left chondrocostal joint, it represents the tricuspid valve, which is actually higher and to the right of the sternum. The tricuspid focus represents the outflow tract of the tricuspid valve and the direction of sound transmission that follows valve closure.

Mitral or apical focus. Located in the fifth left intercostal space, in the midclavicular line, it represents a valve and a cardiac chamber. Blood flows from the mitral valve, which is higher and to the right of the mitral or apical focus. The tip of the left ventricle also lies behind this focus, and a pulsation can be palpated as the ventricle contracts.

There are additional areas or focuses that, due to their usefulness, should also be examined:

Sternoclavicular area. It rests on the sternum and its junction with both ribs, as well as part of the first left and right intercostal spaces. These structures can serve as demarcation lines to evaluate the aortic arch and pulmonary artery, which are located to the left of the first intercostal space.

Right ventricular area. Above the right ventricle that faces the anterior thorax, it extends from the third intercostal space to the distal end of the sternum. The right lateral border of the area is located above the right atrium. The left ventricle is on its left lateral border.

Erb focus or second aortic focus. In the left third intercostal space it is included in the right ventricular area. It is considered by most to be the fifth main focus of precordial auscultation. Aortic and pulmonary valve sounds, primarily murmurs of aortic insufficiency, may be transmitted to this point.

Ectopic area or mesocardium. It represents the demarcation line where abnormal precordial pulsations can be palpated in people with disorders of the left ventricular wall, secondary to angina or diffuse myocardial disease. Typically such pulsations can be detected in the left middle precordium, just above the left ventricular apex. However, this location varies between people.

Epigastric area. It represents the same anatomical region for both abdominal and cardiac examination. Aortic and right ventricular pulsations can be detected in this area.

CLINICAL EXPLORATION

The examination will be carried out using the four classic procedures that we already know: inspection, palpation, percussion and auscultation.

Inspection and palpation usually precede percussion and auscultation. However, it may sometimes be useful to simultaneously perform inspection-palpation, inspection-auscultation, or palpation-auscultation, especially when abnormal findings are observed. For example, if an abnormal pulsation is detected in the neck, by inspection and/or palpation, it is useful to auscultate while palpating the pulse or inspecting the neck veins, to know where the pulsation falls within the cardiac cycle.

It is important to note whether the examination findings occur during early, mid, or late systole or diastole, as well as whether they occur intermittently or continuously. Also describe if there is any variation of the findings with respiratory moments.

INSPECTION

Observe from the right side, although the inspection can be carried out on either side. A view from the foot of the bed may be helpful. Through it, observe all the visible phenomena: skin color, architecture of the region and external configuration, through static inspection, and heartbeats, through dynamic inspection, whether positive heartbeats in the direction of elevation, or negative heartbeats in the direction of depression, both in the precordial region and in the epigastric and cervical regions.

Dynamic inspection

It allows us to appreciate the "tip beat" or "apexian beat", inappropriate terms according to some authors, since they do not correspond to precise anatomical or clinical concepts, which is why it is preferable to refer to the tip shock. Also inspect the existence of other beats unrelated to the tip shock.

tip shock

William Harvey (1628) in his work, The Circulation of the Blood, dealt with the tip shock and destroyed many of the erroneous ideas that existed about its origin.

Concept

The lifting that the apex region experiences, due to the push of the tip of the left ventricle forward, during the beginning of cardiac systole, is called a tip shock, which is why it is also called the point of maximum impulse (PMI).

Production mechanism or semiogenesis

Tip impingement is a complex phenomenon generated by the contraction of the heart chambers, and lasts only part of the contractile period. The following mechanisms intervene in its production:

1. Atrial systole causes a small elevation when the ventricles suddenly fill, which precedes the elevation produced by these cavities when they contract.

2. Ventricular contraction considerably increases the consistency of the organ, which allows the chest wall to be pushed.
3. The heart rotates around its longitudinal axis and, as a result, the apex moves forward and inward, coming into contact with the precordial wall.
   This rotation appears to be small, as revealed by recent imaging examinations.
4. When contracting, the heart changes its shape, it becomes more spherical, thus increasing its antero-posterior diameter.

5. There is also a decrease in the size of the heart, as its ventricles suddenly empty during the expulsive period.

6. A vacuum is also produced in the vicinity of the heart, due to the decrease in its volume, with a change in shape, and due to the flow of blood out of the thoracic cavity; This void tends to be compensated quickly thanks to the expansion of the lung flaps and the entry of blood into the thorax.

7. Finally, there is a backward movement of the heart forward, downward and to the left, due to the sudden ejection of blood into the large arteries.

In summary, it is accepted that the change in consistency and shape of the ventricles, as well as the recoil movement of the heart, seem to be the most important elements in the genesis of apex impingement in normal and pathological conditions.

Clinical characteristics or semiography

Inspection of the tip shock allows us to determine its situation or location, shape, intensity, extension, frequency and rhythm.

Situation

The outermost and lower point where the heartbeat is observed should correspond to the tip of the heart.

Normally in adults, the impingement of the tip is found in the fifth left intercostal space (fourth in children, sixth in the elderly) above the midclavicular line (MCL) (mitral focus) or a little further inside, limited to covering a reduced extension, which covers approximately the tip of a finger, and which should not be larger than 2-3 cm in diameter.

Tip impingement may not be visible in all people. It is more easily visible in thin individuals than in thick ones, because the exaggerated development of the adipose panniculus or pectoral muscles makes its observation difficult, to such a degree that it can be missing in such conditions. It can be observed better in men than in women, since female breasts make it difficult to appreciate, especially in those who have exuberant breasts. However, always try to observe the apical beat in women, asking the person to lift their left breast or doing so gently with the thumb of your left hand, while tangentially observing the inframammary fold, which should correspond to the fourth-fifth left intercostal space, in search of the apical beat.

The situation of the tip impingement undergoes variations in physiological state, according to the constitution, abdominal pressure and position of the subject. In pynics, in obese people and in pregnant women, during the last stages of pregnancy, the beat of the tip rises and can be observed above the fifth intercostal space and somewhat outward, because the diaphragm moves upward; while in the long and thin ones, it is located lower and inward. In left lateral decubitus (Pachón position), the tip deviates 2-5 cm towards the axillary region; The right lateral decubitus position moves it less towards this side.

The physiological alterations of toe impingement are best observed with the individual seated.

Other times, usually in pathological situations, instead of a lift, a depression of the apical region occurs.

Form, intensity and extension

The intensity of the tip shock depends, on the one hand, on the thickness of the wall, and on the other, on the size of the heart and the strength of its contraction.

Frequency and rhythm

It is much easier, and safer, to determine heart rate and rhythm by auscultation than by tip shock inspection. However, observing the frequency of the tip shock and whether it is rhythmic or not is already an advance to specify these aspects in greater or lesser depth during auscultation.

Other beats unrelated to the tip shock

If you find another beat unrelated to the tip shock, also describe its location or situation and its shape, intensity and extent.

There are other beats unrelated to the tip shock that can also be positive or negative. Under normal conditions, the epigastric beat can be observed; In thin individuals, after ventricular systole, the forward projection of the epigastric region occurs, causing the heartbeat. In healthy subjects, cardiac erethism due to physical exertion, emotion, etc., favors its appearance.

Other times, the epigastric beat consists of a systolic contraction of that region, a negative beat, motivated by the transmission of the heartbeat, which can happen in the physiological field; However, such a finding may be abnormal and should be evaluated later, because it occurs more often when the right ventricle hypertrophies.

To discover these beats it is almost always necessary to resort to tangential inspection, but in practice their presence is complemented and specified by simultaneously using palpation.

Since beats other than tip shock are usually abnormal, their study will be discussed in more detail in Section II.

PALPATION

Exploration technique

With the person supine, palpate the entire precordial area with the palm of the hand. The palpating hand (whose temperature must be moderate) will be applied flatly, first covering the mesocardium and the tip; then the xiphoid region and its surroundings, and finally the base, on both sides of the sternum, placing the hand transversely and palpating the upper segment of the sternum and the adjacent parts on the two sides of the thorax.

To specify certain palpatory phenomena and above all to locate their extension, we resort to

Therefore, limited palpation with the fingertips.

The palm of the hand is more sensitive to vibration.

The fingertips are more sensitive to pulsations, such as tip shock.

While palpating, you can position the stethoscope slightly to place the findings in time with the cardiac cycle.

Palpation must be performed in different positions:

supine; with the individual seated; in left lateral decubitus, to better perceive apex phenomena; sitting with a slight inclination to the left, for the best perception of the basals, and, sometimes, in ventral recumbency (fig. 11. 2).

The existence of pain in the precordial region upon palpation with the tips of the fingers must be ruled out, which is generally due to osteomyoarticular or neurological causes, rather than cardiovascular alterations, but which is always pathological.

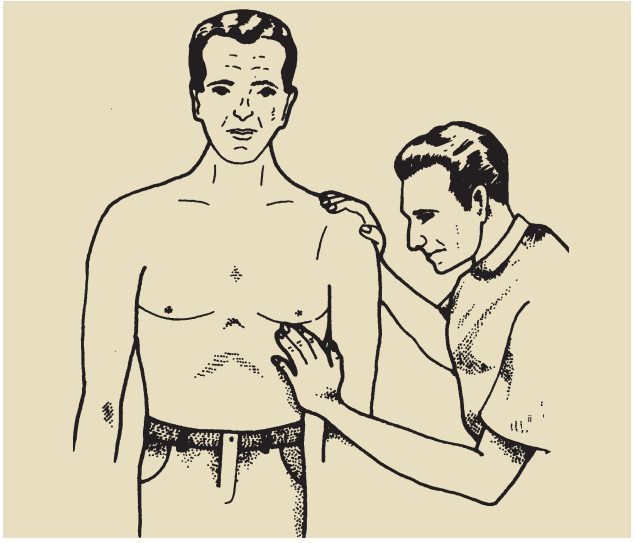
Palpate the precordial region in search of the most important elements that can be obtained by palpation, which we will describe below:

- Pulsatile movements (tip shock and others).
- Palpable valve vibrations (valvular shocks).
- Catharic shudder (fremitus or thrill).
- Diastolic gallop rhythm.
- Palpable pericardial rubs (pericardial fremitus).

Palpation of any of these elements must be subsequently described in terms of location, amplitude, duration and direction of the impulse.

Pulsatile movements. Normally only the apex pulse or apex impingement can be palpated over the mitral focus.

Fig. 11.2 Foot palpation of the tip impingement, with the tips of the fingers.



tip shock

Physiological variations

In children and young people it is common to perceive the heartbeat in the supine position; while in adults over 30 years of age, it is common not to find any palpable heartbeat in the supine position.

Dressler states that palpating it in that position when the subject is over 30 years old should raise the suspicion of some cardiac alteration. On the other hand, in left lateral decubitus, its verification is constant, except in cases of dextrocardia.

Therefore, when tip impingement is not identified in the left hemithorax, in left lateral decubitus or in dorsal decubitus, it is necessary to look for it in the right hemithorax, even resorting to right lateral decubitus.

As the rest of the pulsatile movements and the other elements that can be obtained on palpation are generally pathological, in this Section we will only give the most general elements of some of them.

Catary shudder (fremitus or thril)

Concept

It is a sensation perceived by the palpating hand, comparable to the sensation obtained by palpating the back of a purring cat against the grain.

It is proposed that it is produced by the regular succession of a series of fine vibrations that come from the heart and vessels and are transmitted to the chest wall; Some of these vibrations are also transmitted to the vessels of the neck.

In normal hearts, particularly in subjects with cardiac erethism (young, sympathicotonic, hyperthyroid, etc.) and thin chest wall, a vibrating sensation similar to thrill can be perceived during systole, which is called tremor cordis. Tremor has no pathological significance.

Production mechanism

The production mechanism is similar to that which causes heart murmurs. When the blood flow passes from a narrow portion to another of greater amplitude, fluid vortices are formed that, when they collide with the cardiac or vascular walls, make them vibrate, producing the sensations already described. The thrill is, therefore, the tactile manifestation of a breath.

Exam technique and thrill characters

To investigate the presence of thrill, it is preferable to palpate the precordial region with the whole hand. As explained previously, vibrations are more easily identified if we palpate with the palmar portion.

corresponding to the metacarpophalangeal joints, which we explore with the tips of our fingers.

Catary shivers can occur during systole, in full diastole, or cover both times, without interrupting, receiving the name: systolic, diastolic or continuous thrill (systodiastolic). Its location is variable; They can be located at the tip, at the base, in the vessels of the neck, in the mesocardium or, exceptionally, in the xiphoid region.

The apex thrill is usually better perceived in the left lateral decubitus position, while the basal thrill is more easily externalized when ordering the subject to sit up if he is lying down; If he is sitting, ask him to lean his trunk forward and to the left.

Sometimes, in the case of basal or xiphoid thrills, ventral recumbency is better. It is usually best for the pressure exerted by the palpating hand to be light; although, other times, the sensation is obtained more easily by pressing harder on the region being explored.

The pathological alterations of the palpation of the tip beat, the semidiagnosis of the thrill according to its semiography and the rest of the palpable elements, which are always abnormal, will be described in the Propedeutics Section. Combined inspection and palpation in the epigastric region and the cervical region are used when we want to delve deeper into abnormal findings, which is why they will also be described in said Section.

PERCUSSION

Cardiac percussion is a very subjective method and prone to error. Although theoretically it seems that it should not offer any technical difficulty, since the percussive qualities of the heart (matte sound) and the lung (tympanic sound) are different, in reality it happens in a very different way.

In addition to the inconvenience represented by the curvature of the thorax, the development of the adipose panniculus and the muscles, the greater or lesser resonance of the rib cage, the breasts in women, etc., it must be taken into account that the systolic rotation of the heart reduces its thoracic projection area by more than 1 cm: without analyzing the criteria of those who claim that anyone who wants to design the heart with percussion wastes their time... and deceives themselves. Likewise, we believe that this method, despite recognizing that the data obtained do not faithfully correspond to the dimensions of the organ, deserves to be studied and explored.

General considerations

The anterior face of the heart is partially covered by the lung flaps, so that there are two Zones: one that corresponds to the part that is in direct contact with the chest wall and another that is separated from it by lung tissue. These areas have been denominated respectively, zone of absolute dullness and zone of relative dullness, because the first produces a dull sound and the second a sound intermediate with that of pulmonary sonority, which is identified as submatt.

Relative dullness

The right border of the zone of relative dullness is normally given by the right atrium (between the third and fifth intercostal spaces) and higher up by the initial portion of the ascending aorta. The left border is formed by the left ventricle downwards and the cone of the pulmonary artery upwards.

Exploration technique

The technique to delimit said area is as follows (fig. 11.3):

a) Determine the upper border of the hepatic dullness, beginning to strike from the infraclavicular region towards the base of the thorax.

b) Determine the right border of the cardiac area by striking transversely from the right anterior axillary line towards the sternum, at the level of the third, fourth and fifth intercostal spaces.

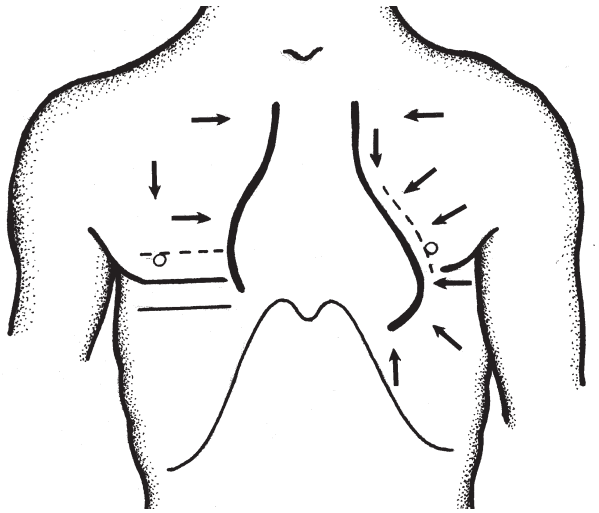
c) Determine the left border by striking in a transverse and oblique direction from the left anterior axillary line towards the sternum, and also in an ascending or descending vertical direction.

d) Direct the patient to perform several forced expirations and mark each limit with a dermographic pencil until said area is configured.

Absolute dullness

The area of normal absolute dullness has the shape of a triangle whose vertex is at the level of the fourth face.

Fig. 11.3 Percussion: relative dullness.



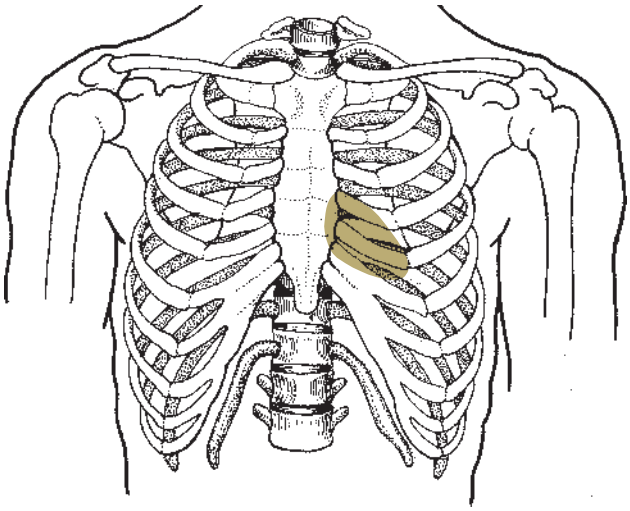


Fig. 11.4 Percussion: absolute dullness.

costal tilago and whose base is confused without delimitation with hepatic dullness. The vertical right edge corresponds to the left edge of the sternum and the left edge is oblique downward and outward, extending from the vertex to a little inside the impingement of the tip (fig. 11.4). This area of absolute dullness is produced by the right ventricle in normal subjects.

Semiotechnics

The percussion will be done with a weak percussive blow, marking only the points where the sound is dull; The technique to follow is:

a) To strike the right edge, the pleximeter finger is placed in the direction of the longitudinal axis of the thorax at the right limit, which we determine in the relative dullness by advancing transversely to the left until absolute dullness is found. This operation is carried out at the level of the fourth and fifth spaces.

b) To determine the left edge we do a similar operation but in the opposite direction, always starting in parallel from the left limit of relative dullness.

c) It corresponds to what is stated in the relative dullness subsection.

AUSCULTATION

Auscultation is one of the most valuable clinical methods for examining the heart, especially for the diagnosis of valvular conditions that can be recognized by this means before they have caused changes in the shape and size of the heart and, consequently, the electrocardiogram. serves also to recognize the stages of myocardial claudication and cardiac rhythm alterations. It constitutes an exploration method whose advantages cannot be matched by any other.

General considerations

Sound. Some of its properties

In order to correctly develop cardiac auscultation, it is essential to previously specify some concepts about the production, characteristics and transmissions of sounds, more closely related to this topic.

The ear perceives sound when vibrations of certain frequency and intensity reach it. In every acoustic phenomenon three qualities are distinguished: tonality or tone, timbre and intensity.

Tonality or tone

The pitch of a sound is determined by the number of vibrations per second. The lower the frequency, the deeper the sound. If the frequency is less than 20 vibrations per second or greater than 20,000, the human ear does not perceive any sound. Many of the acoustic phenomena of the heart have a tone that is below the threshold of human hearing. About 80% of the energy of the first and second heart sounds is at frequencies less than 70 vibrations per second, and most sounds and murmurs have less than 500 vibrations per second. Components with frequencies above 650 vibrations per second are of little importance in auscultation. There are personal differences in the ability to perceive sounds above or below the common limits of hearing. The ability to perceive low frequency sounds is very advantageous and can be acquired with proper training.

Ring

In addition to the fundamental vibrations that determine pitch, most sounds have higher or lower frequency vibrations, called overtones, that determine the timbre of the sound. Thus, the same note has different overtones, depending on whether it originates from a piano, violin or trumpet; This difference in timbre is what allows us to recognize the note coming from each instrument.

instrument.

Intensity and strength

Intensity refers, strictly speaking, to the physical aspect of the sound; while force, to the subjective component of this.

The intensity of a sound is proportional to the amplitude of the vibration and is independent of hearing.

The strength corresponds to the degree of sensation produced, and depends on both the intensity of the sound and the sensitivity of the ear to that particular sound.

The ear is most sensitive to sounds whose frequency ranges between 500 and 5,000 vibrations per second. Below 500 vibrations its sensitivity decreases rapidly. Therefore, a sound of 500 vibrations per second will be heard louder than one of 100, even though both have the same intensity. Since many heart sounds have frequencies less than 100 vibrations per second, and most have frequencies less than 500 vibrations, when auscultating, the ear is practically at the limit of its hearing threshold.

Although force and intensity do not have the same meaning, in general and also in this book, the term intensity will be used in the same sense as force.

Associated with intensity is the phenomenon of masking, which consists of the reduction of the ear's ability to perceive certain sounds in the presence of others. The ear adjusts to the intensity of the sound it is perceiving. A loud sound induces you to protect yourself by reducing your perception acuity. If this loud sound is immediately followed by a weak one, the ear will not be able to pick it up. This phenomenon is why soft murmurs that follow loud noises are difficult to hear or are not heard at all.

The same thing happens if weak noises follow loud noises.

Another type of masking occurs when a complex sound (made up of tones of different frequencies) undergoes an increase or decrease in intensity.

The masking of some of the tones by others varies with intensity and can occur at any given level of intensity. The timbre of a sound can be modified by varying the intensity, depending on whether certain tones are no longer perceived or reappear, due to the masking effect. When a murmur spreads to a distant point on the chest, decreasing its intensity, its timbre can vary in such a way that it is possible to doubt the identity of the transmitted murmur.

In addition to tone, timbre and intensity, sounds have duration. They can be long or short. The relationship between spaces and noises allows the location of cardiac acoustic phenomena.

Transmission

Noises originating in the heart or its surroundings must be transmitted to the chest wall and from there to the ear in order to be perceived. We will describe below some of the factors related to this transmission:

1. Estrangement. As a sound moves away from the source of production, its intensity decreases depending on tion of the square of the distance to the source. Generally the noises are louder in the area of the chest closest to the point where the noise occurred.
2. Reflection. As the sound is transmitted towards the chest wall, it is modified by reflections that occur when the medium it passes through varies; for example, from the heart to the neighboring muscles, the chest wall or the lungs, etc. When a sound passes from one medium to another, some of it is reflected and the rest passes through the medium. Although there are many factors that influence the value of the reflection, the most important is the difference in density between the limiting tissues. If the medium is relatively homogeneous, most of the sound passes through it and only a small portion is reflected; The opposite occurs if the densities of the limiting media differ greatly from each other. Blood and muscle tissue, of similar densities, transmit sound without much reflection.

   The lung, on the other hand, due to its air cavities, has a lower density, and the sound that must pass from the muscle to the lungs, and from these to the chest wall, suffers a large number of reflections that prevent its correct transmission. Because of the great difference in density that exists between the chest wall and the air, only the loudest sounds can be heard by immediate auscultation.

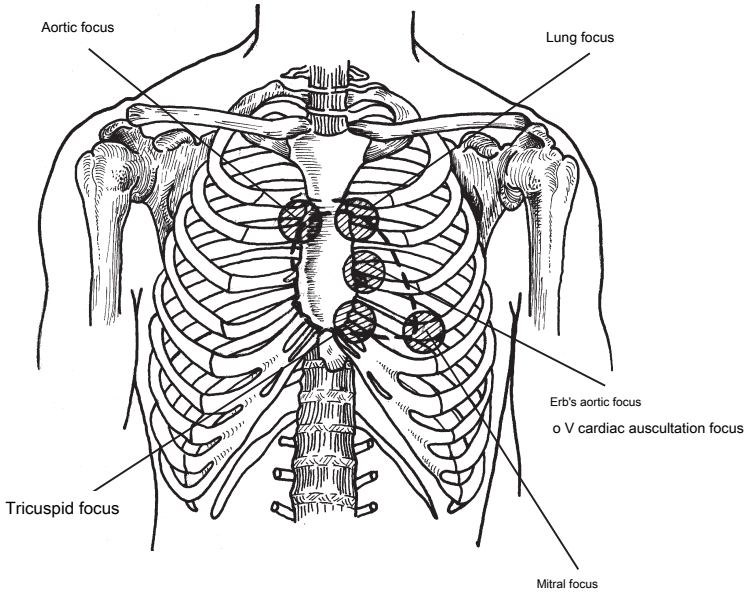
3. Friction. When a sound passes through a medium it suffers a loss of intensity, which is due to friction.
   This does not affect all frequencies equally; Therefore, the sound, in addition to losing intensity, can change its timbre since this depends on the frequencies of the different components of the sound. Thus, as the same murmur is followed over different areas of the chest, its timbre may vary. This change and those resulting from masking must be taken into account if trying to determine the existence of two murmurs or a single propagated murmur, based on the timbre heard in different areas of the chest.

Regions where auscultation should be practiced

Auscultation should be performed throughout the precordial region, both in its four main auscultation foci: tricuspid, mitral, pulmonary and aortic (Fig. 11.5), as well as in the additional areas and foci of the precordium (second aortic focus or fifth auscultation focus and mesocardium) and outside of it, such as the region of the base of the neck, the sternoclavicular area, and the epigastric, especially the region below the left costal margin, near the xiphoid appendage, where the right ventricle projects precisely.

Auscultation in the epigastrium sometimes allows us to collect phenomena that auscultation of the precordial region had not revealed, since in the latter, acoustic phenomena

Fig. 11.5 Cardiac auscultation foci.



Cardiac problems may be hindered by the subject's obesity, or by emphysema, and are not perceived clearly enough.

Auscultation in the neck region allows us to know if the auscultatory phenomena that we hear in the precordial region are perceived in this region and, in addition, to collect some signs that depend on the blood vessels that are there. The aorta can sometimes appear behind the fork of the sternum, and the subclavians, above the clavicles.

Finally, to study some characteristics of heart sounds and murmurs and their propagation, other thoracic areas must be examined in the anterior plane of the thorax and the lateral planes, especially the left lateral plane.

Remember that the heart is not all in contact with the chest wall, but in certain regions; that blood flow transmits sound beyond the point of its origin; and that the vibratory phenomena that originate in the valve or in a chamber must reach the costal wall, where we hear them precisely being transmitted through the ventricular or arterial walls.

Heart sounds originating from the valves are detected in the direction of blood flow in one of the four main areas or foci, where the heart and great vessels come into closest contact with the chest wall.

When auscultating the precordial region we must follow a systematic sequential method, always the same, and to which we must become accustomed. We can begin with the tricuspid focus, which, as we will see later in Sec. tion II, it is rarely affected, and therefore, the most normal heart sounds are recorded in it. After the tricuspid focus it passes to the mitral focus; then, to the foci at the base: the pulmonary and the aortic, and to close the circuit of the foci, one must move to the second aortic focus. Afterwards, the entire precordial region is covered, since in the mnesocardial area there will be very important data as in the other regions that we have mentioned. It must always be done in the same way so that we automatically listen to all these sites.

Another sequential method may be to start with the aortic focus, where the first and second sounds are very well identified, and then auscultate the vessels of the neck or move to the pulmonary focus. Then it descends along the left sternal border, to the second aortic focus, to the tricuspid and the epigastric region, to end at the mitral focus and nmesocardium.

It is also recommended by Levine to auscultate in neighboring and nearby areas until the area is covered (systemic).

Systematic auscultation

With the person supine, proceed systematically from one focus to the next.

Auscultate each area using the diaphragm to detect higher-pitched sounds, such as the first sound (R1), the second sound (R2), and the murmur of aortic insufficiency.

Use the bell to detect the lowest sounds, such as the third and fourth sounds (R3 and R4).

Also listen to the base of the heart with the person sitting leaning forward and then evaluate the apex with the person in the left lateral decubitus position.

Listen for several cardiac cycles at each auscultatory focus.

I. Determine heart rate and rhythm

To evaluate heart rate and rhythm, it is not necessary to auscultate all precordial sites, because the sound is transmitted well and the frequency and rhythm will not vary. So in the first focus of the sequence, determine these two elements.

The rhythm will usually be regular, but it can vary in some people, especially in children and young adults, in whom an irregular rhythm that varies with breathing can be seen. During inspiration, venous return is greater and the frequency may increase to compensate for the greater blood volume. The frequency will then decrease with expiration. This is called respiratory sinus arrhythmia and has no pathological significance.

If from the beginning of the auscultation or at any time during it, the heartbeats are not rhythmic, you should evaluate this aspect in detail later.

If the beats are not completely rhythmic, but you can specify a basic rhythmic cadence, where premature beats are inserted followed by a pause (extrasystoles), this is an extrasystolic arrhythmia. If the auscultated beats are completely arrhythmic, without requiring a base cadence, it is a complete arrhythmia, which is always pathological and is usually due to atrial fibrillation.

When an arrhythmia is detected, the examination should be completed by auscultation simultaneously with palpation of the radial pulse. Extrasystoles or premature beats can be ventricular, if they originate at this site, or supraventricular, if they originate above the same site.

mo.

Usually the ventricular contraction of supraventricular premature beats occurs at times when there is already enough blood in the ventricles to be expelled to the periphery and the premature beat reaches the radial pulse, so the extrasystole and its subsequent pause are detected simultaneously with cardiac auscultation and pulse palpation.

When the extrasystole is of ventricular origin, premature ventricular contraction occurs when the left ventricle does not have enough blood to expel to the peripheral arteries and the extrasystole is auscultated, but it is not transmitted to the radial pulse and palpation of this produces the sensation that a beat is missing.

In summary, supraventricular extrasystoles are generally transmitted to the pulse, while ventricular extrasystoles are not transmitted, and the pulse is palpated as the absence of a beat.

Complete arrhythmia due to atrial fibrillation is confirmed by simultaneous palpation, because all arrhythmic beats reach the pulse, that is, they are heard and palpated synchronously.

Heart rate is determined by counting the beats in one full minute, with a watch that counts the seconds, while listening. It can be left until the end of the auscultation, but it tends to be forgotten. The normal resting rate is 60-100 beats/min, but it may be lower in people in good physical condition.

II. Identify heart sounds in each focus

Auscultation of the heart reveals the presence, in each cardiac cycle, of two sounds in relation to the closing of the valves of this organ. In some normal young people, a third sound of uncertain origin may also be heard.

Between the first and second noises there is a very brief pause, called first silence or small silence, and between the second and first noises of the following cycle, a second pause of longer duration, called second silence or great silence.

Listen to each noise separately and try to block out the rest of the sounds. Specify its intensity, tone and if any of it is doubled.

First noise (R1)

The first heart sound (R1) is slightly low in pitch and has a slightly longer duration (0.14 s) than the second heart sound. Its onomatopoeia is dom. It has greater intensity at the tip when heard with the diaphragm, where it is heard as a single sound.

The factors in the production of the first heart sound are:

1. Simultaneous closure of the atrioventricular valves, at the beginning of ventricular contraction. This is the fundamental factor.
2. Muscular factor, derived from ventricular contraction.
3. Arterial factor, originating from the vibrations produced by the systolic distension of the aorta and pulmonary artery.
4. Atrial factor, determined by the preceding atrial systole.

In practice R1 represents the closure of the mitral and tricuspid valves and marks the beginning of ventricular systole.

The silence that mediates between the first and the second noises, or small silence, is a systolic space; so that any superadded noise will have this hemodynamic connotation, that is, it will be systolic.

Second noise (R2)

The second heart sound is slightly higher pitched and shorter (0.11 s). Its onomatopoeia is lop. It has greater intensity in the foci at the base. In young adults it can normally be perceived unfolded at the end of inspiration.

The determining factor of the second heart sound is the simultaneous closure of the aortic and pulmonary sigmoid valves, the beginning of ventricular diastole.

The silence that intervenes between this noise and the first of the next cardiac cycle represents a diastolic space and any phenomenon that occurs in this period will be diastolic.

Note that in the normal heart the closing of the valves produces noise, while the opening is silent.

In a person with normal rhythm and frequency, the first and second sounds will be immediately distinguishable, due to the shorter time interval between RI and R2. R1 can also be heard almost simultaneously with palpation of the carotid pulse shock, which may help identify R1 and R2 in people with high heart rates.

Third noise (R3)

It occurs shortly after the second noise (0.13-0.18 s).

It is believed to have its origin in vibrations of the ventricular wall that result from the impact of the blood flow that enters during rapid ventricular filling. Due to its low intensity and low tone, it is not commonly heard, although it is recorded with some frequency in phonocardiograms. It requires a meticulous search, for which the bell is used. In children and young people it is heard with some frequency at the tip level. It usually disappears after 25 years of age.

When it appears at later ages it is always pathological and in this case indicates the existence of ventricular failure; Thus a three-beat rhythm is created, a prelude to a gallop rhythm.

II. Identify the normal splitting of R1 and R2 Normal splitting of RI

As the left side of the heart normally contracts before the right, the mitral valve (M) closes before the tricuspid valve (T) and a first sound (R1) is produced, split into two components (M1 and TI), which are separated from each other by 0.02-0.04 s. However, the time interval may be too short to differentiate these components.

The normal splitting of R1 can be heard next to the tricuspid area or at the left lower sternal border and as we get closer to the mitral focus it is less necessary, so that RI is heard as a single noise at the tip. There are no variations in RI with respiration.

Normal splitting of R2

Also called physiological splitting, it can be heard on inhalation.

Inspiration increases venous return to the right side of the heart. At the same time, pulmonary vascular capacity increases and more blood accumulates in its bed, which decreases the amount of blood entering the left heart.

As a result, the left ventricle empties faster than the right and the aortic valve (A) closes first than the pulmonary valve (P), around 0.04 s, and gives rise to an R2 normally split into two components (A2 and P2). On expiration R2 is heard once again.

IV. Identify abnormal heart sounds and abnormal heart sounds

Once R1, R2, and any normal splitting have been identified, look for abnormal splitting of RI and R2 by listening with the diaphragm and concentrate on identifying any other extra sound, which is measured in time in relation to the cardiac cycle and respiratory phase.

Abnormalities of heart sounds and accessory sounds will be studied in Section II.

V. Identify the presence of murmurs and, if they exist, describe their characteristics

Generalities of murmurs

They are the noises that appear in relation to the cardiac cycle in the precordial region or in its vicinity, with acoustic characteristics that have been compared by Laenec to the noise of a bellows fanning the fire.

Its semiological importance is considerable, because in addition to murmurs that show a valvular heart lesion, there are other innocuous murmurs that, if misjudged, can lead a healthy subject to be considered as having a heart disease, with the limitations that this entails. Gasue affirms that the murmur is actually the greatest producer of iatrogenic disease that medical science and its instruments have created.

The murmurs, for the most part, are produced by a turbulent flow. The occurrence and degree of turbulence depend on the speed and viscosity of the blood, and the type and configuration of the obstacle that arises in the bloodstream.

Criteria for describing murmurs

The evaluation of a murmur includes the description of eight general characteristics: its intensity, tone, timbre, the moment of the cardiac revolution in which it occurs, its duration, the place where it is heard most intensely, its propagation or irradiation and the modifications.

nes that the murmur experiences under the influence of breathing, muscular effort, changes in position and treatment.

1. Intensity. How intense is the murmur? To describe its intensity use the following degree system:

Grade 1. Very weak. Poorly audible.

Grade 2. Weak. Audible only in silence.

Grade 3. Moderate. Clearly audible.

Grade 4. Intense. It can be associated with thrill.

Grade 5. Very intense; palpable thrill. It can be heard with the stethoscope partially outside the chest.

Grade 6. Very intense, maximum intensity. Palpable Thrill, you can hear it, even without the stethoscope.

There are other scales with fewer degrees, so it is advisable to clarify which scale is being considered. For example, if a murmur considered grade 2 on this scale of 6 is detected, it is expressed and recorded as 2/6 (two out of six).

Intensity also includes the way a murmur goes from minimum intensity to maximum intensity, or vice versa.

versa.

Increscendo or growing.

Decrescendo.

- Rhomboidal or diamond increscendo-decrescendo. When the murmur increases until it reaches its maximum intensity and then decreases progressively until it disappears.

2. Tone. According to what we explained previously in the generalities of sound, it can be: high or sharp; low or serious.

3. Doorbell. It is associated with a known quality of another sound. Examples: soft or aspiration, blowing, scratchy or rough, in machinery, steam jet, musical, etc.

4. Time in which they occur. It refers to the moment of the cardiac revolution in which they occur. Does it occur in systole, diastole or is it systodiastolic?

Diastolic murmurs are always produced by organic damage to the valvular apparatus. Systolic murmurs can be organic or functional.

5. Duration. Describe the exact time in relation to the cardiac cycle as follows:

Pansystolic (holosystolic), It occupies the entire systole, the entire space of the small silence, between the first and second sounds and generally masks the latter.

Holodiastolic. It occupies the entire space of the great silence, between R2 and the RI of the next cycle.

Protosystolic and protodiastolic. It occurs early in systole and diastole, respectively.

Midsystolic (ejection). It begins after hearing R1, peaks in midsystole and ends before hearing R2.

Mesodiastolic. It occurs in the middle of diastole.

End systolic. Part of the small silence is heard and the murmur begins immediately before the second noise is heard, late in systole.

End-diastolic or presystolic. It occurs late in diastole, immediately before the RI of the next cycle.

6. Location. It is the focus or place where it is heard with greater intensity and clarity, which allows us to deduce the valve device or the chamber that produces it.

7. Propagation or irradiation. Once the site of greatest intensity has been determined, irradiation is the site(s) to which the murmur spreads, according to the direction of blood flow, when it occurs.

8. Modifications with position, ventilation, exercise and treatment. Of great importance to complete the necessary elements in diagnosing the cause of a murmur.

These eight characters will be studied more widely in the chapter dedicated to murmurs, in Section II.

Anorganic or accidental tip murmurs

They are observed with great frequency.

Characteristics:

- Intensity: grade 1-2/6, therefore its intensity is low.

- Tone: variable.

- Timbre: variable.

- Moment of cardiac revolution: systole.

- Duration: they are always merosystolic, they do not cover the entire small silence, therefore both heart sounds are heard; His onomatopoeia will be: dom-fut-lop.

- Site of greatest intensity: auscultation area of the tip.

- Propagation: the propagation of these murmurs is null and, therefore, they are heard exclusively at the tip.

- Modifications: these murmurs change with the patient's position, with respiratory movements and from one day to the next.

Accidental core blow

The accidental systolic murmur at the base of the heart, to the left of the sternum, perceived at the level of the pulmonary focus, the second left intercostal space next to the sternum, is one of the most frequent accidental murmurs. Due to this last characteristic, many authors have considered it a physiological murmur, that is, it is a normal phenomenon, which does not correspond to a pulmonary valvular or vascular injury.

Characteristics:

- Intensity: up to grade 3/6.
- Tone: variable.
- Doorbell: soft.
-Location: systolic.
- Duration: merosystolic, that is, they occupy only a small part of the small silence.
- Site of greatest intensity: pulmonary focus.
- Propagation: does not spread.
- Modifications: it varies with respiratory movements, with changes in position and with the days.

Auscultation of the pericardial rub, venous rumor and extracardiac arterial murmurs will be studied in the chapter dedicated to murmurs in Section II.

EXAMPLE OF THE WRITTEN RECORD OF THE EXAMINATION OF A NORMAL PRECORDIO

Inspection. Beat of the tip visible in the fourth intercostal space, in the CML. No deformities or other pulsating movements are observed.

Palpation. Impact of the tip palpable in the same visible place. No other pulsating movements, fremitous thrills, or friction are palpable.

Percussion. Percussable cardiac area within normal limits.

Auscultation. Heart sounds normal, rhythmic and of good tone and intensity. No accessory noises, murmurs or rubbing are heard. HR: 80/min.

EXPLORATION OF THE PERIPHERAL VASCULAR SYSTEM. PERIPHERAL PULSES AND BLOOD TENSION

12

The examination of the peripheral vascular system (PVS) includes the exploration of:

1. Peripheral arterial system:
   a) Arterial pulses.
   b) Radial pulse frequency (PR).
   c) Measurement of blood pressure (BP).
2. Peripheral venous system:
   a) Venous pulses.
   b) Alterations of the venous vessels.
   c) Venous pressure.
3. Alterations of the lymphatic vessels.

In this chapter we will not refer to SVP alterations, which will be addressed in Section II, Chapter 47.

PERIPHERAL ARTERIAL SYSTEM

The examination of the arterial pulse and that of blood pressure or tension constitute two elements of capital importance in the physical examination of any subject and especially of those who suffer from cardiovascular conditions.

PULSE STUDY

Concept and production mechanism

The pulse is a wave determined by the sudden distention of the walls of the aorta, caused by ventricular ejection, which spreads to the arteries thanks to their elasticity. The propagation speed is 8-10 m/s, so that the wave reaches the arteries furthest from the heart before the ventricular evacuation period has ended.
This speed increases as arterial elasticity decreases, which is why it is greater in the elderly than in the young.

Examination of arterial pulses

The examination of these pulses logically begins with inspection, looking for visible pulses, which are pathological, so arterial pulses are generally evaluated by palpation, with the tip of the fingers.

fingers, in places where the wall of an artery can be compressed on a bony or hard plane, so that the arterial beat can be felt in the form of elastic rebound of the artery, synchronous with cardiac systole, as pressure is transmitted from the aorta.

The carotid and femoral arteries, as well as the abdominal aorta, should also be heard with the bell and diaphragm of the stethoscope.

Palpation

Degree scale when palpating the pulses

To establish uniformity, pulse findings should be recorded using the following grading system:

Pulse not palpable.

1+ Pulse palpable, but easily obliterated, weak, threadlike.

2+ Pulse weak, but cannot be obliterated.

3+ Easy to palpate, full, cannot be obliterated.

4+ Strong, intense pulse, may be abnormal.

Except for the carotids, pulses should be palpated bilaterally and simultaneously so that useful comparisons can be made.

Palpation sites

Peripheral pulses can be palpated in areas where the large arteries are close to the skin surface. The palpable pulses include, on each side, the temporal, carotid, axillary, humeral or brachial, ulnar or ulnar pulses; radial, femoral, popliteal, posterior tibial and pedis or dorsalis of the foot.

Semiotechnics figs. 12.1 and 12.2)

Pulso temporal. Facing the subject, place your index and middle fingers of both hands over the temporal regions, just above and in front of the pinna, to palpate both superficial temporal arteries, whose beats should have the same amplitude and be synchronous. If you palpate any arterial thrill, you should auscultate these arteries.

Pulso carotídeo. It is the one that most faithfully reflects cardiac functions. Examine each side separately. Place your index and middle fingers in a hook shape, inside the medial border of the sternocleidomastoid, on the lower half of the neck and gently press on the carotid artery. Always palpate below an imaginary line that passes through the upper edge of the thyroid cartilage, to avoid compression of the carotid sinus, which is located at that level, and which produces a decrease in heart rate and blood pressure.

For this reason, this pulse should never be palpated simultaneously.

neously on both sides or very deeply, because if the carotid sinus is pressed, it can result in very hemodynamically significant bradycardias and even more so, in cardiac arrest.

If, in addition to the heartbeat, you palpate any arterial thrill, perform carotid auscultation.

Upper extremities

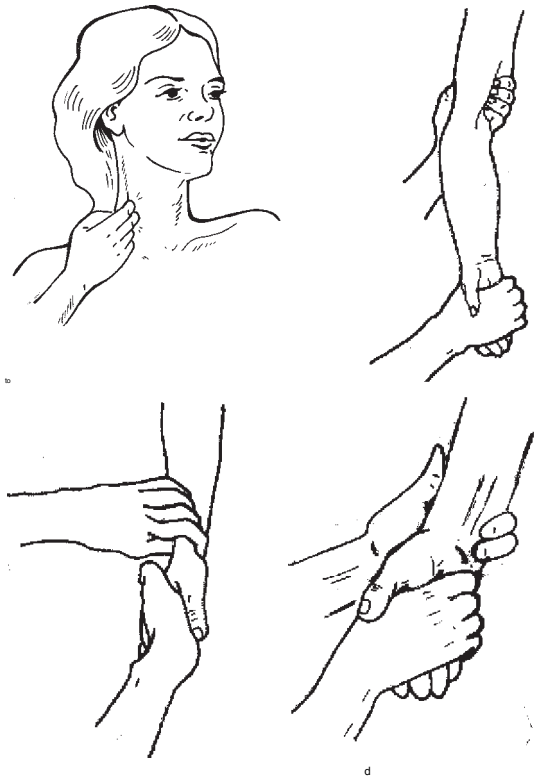
Axillary pulse Raise the arm in external rotation to a 90° angle with the chest wall. Palpate in the axillary hollow, on a line that goes from the midpoint of the clavicle to another located under the insertions of the pectoralis major.

Humeral or brachial pulse. With the subject's forearm slightly flexed over the arm, palpate with your fingers along the medial border of the biceps over the lower third of the arm.

Ulnar pulse. It is palpated on the palmar surface of the wrist joint, above and outside the pisiform bone.

Palpation of the ulnar or ulnar artery is sometimes difficult, but in some clinical situations, such as, for example, when a catheter is placed in the radial artery to monitor blood pressure, this artery must be made evident to ensure adequate collateral blood circulation to the hand.

Fig. 12.1 Palpation of arterial pulsations:
a, carotid; b, humeral; C, radial; d, ulnar.



Radial pulse. The radial artery is the one where it is customary to look for and study the pulse. The examinee's hand is placed slightly inclined inwards and the observer's hand forms a clamp with the three middle fingers on the ventral aspect of the wrist, on the bicipital slide (of the palmaris major), and the thumb placed fell on the dorsal aspect of the wrist.

Lower extremities

In the lower extremities the main arteries investigated are, from bottom to top: the lapedial, the posterior tibial, the popliteal and the femoral (see Fig. 12.2).

Pediatric pulse. It can be located on the dorsum of the foot outside the tendon of the extensor digitorum proprius. Two or three fingers will be used to feel for a pulse. In some subjects it is easy to find; in others, only after a thorough search. Its absence does not necessarily imply the existence of an organic vascular lesion. In fact, it may be absent in 5 to 10% of cases considered normal; On the contrary, a good pedal pulse can be found in atherosclerosis.

Posterior tibial pulse. It should be looked for in the internal retromalleolar canal.

Popliteal pulse. It is palpated in the popliteal region. It is easily found by flexing the leg over the thigh, with the subject in a prone position.

Femoral pulse. It is easy to find at the level of the groin, just at the level of Poupart's ligament or in Scarpa's triangle. In some subjects the artery can be palpated at some distance further down, as far as near Hunter's Channel. Normally the femoral pulse is full and strong.

Fig. 12.2 Technique for palpation of the arteries: a, femoral; b, popliteal; c, pedia; d, tibialis posterior.

Approach to the exam and its registration

1. Ease or resistance to palpation.
   Character of the arterial wall.
2. Synchronism and comparison of its amplitude.
3. Frequency and rhythm of the radial pulse.
4. Pulse hardness, amplitude and contour.
5. Auscultatory findings.

Facilitated resistance to palpation

The arterial pulses should be palpated without difficulty, although they should not be easily obliterated by the pressure of the examiner's fingers. The peripheral arteries, under normal conditions, offer no resistance to the finger that palpates them.

Peripheral pulses, including: radial, posterior tibial, and dorsal pedis, may decrease due to vasoconstriction making them difficult to palpate. A pulse may be difficult to palpate in atherosclerosis, which causes arterial stiffness and decreased elasticity of the arterial wall. Diffuse atherosclerosis can cause pulses to be decreased or even absent. Palpation may be difficult if the skin is edematous.

Non-palpable pulses may also be related to cessation of blood flow and should be evaluated later with Doppler ultrasound.

Character of the arterial wall:

The normal arterial wall will feel soft, depressible, and elastic. The arterial wall hardened by atherosclerosis can be palpated as a rigid cord or a hard, flexible tube, which is sometimes visible, especially in the brachial artery.

Synchronism and comparison of its amplitude

The radial pulse is synchronous and of equal amplitude in both arms in normal individuals. The femoral pulse is also synchronous with the radial pulse.

When we examine a person for the first time, it is necessary to take the radial pulse simultaneously in both arms to compare them with each other, and also compare the synchrony of the radial pulse with the femoral pulse on each side, since there are diseases that can cause alterations in synchronism and pulse amplitude.

We will move on to study the intrinsic characters of the pulse wave, which are explored in the radial pulse: frequency, rhythm, hardness or tension of the pulse, amplitude and Contour.

Radial pulse rate and rhythm Pulse rate:

To determine the pulse rate, it is enough to count the number of beats palpated during one minute at the level of the radial artery. It should not be counted for less than a minute, because sometimes appreciable changes in frequency can occur in this short time.

Physiological variations: in the normal adult the pulse rate ranges between 70 and 80 beats per minute, but 60 to 90 minutes can be accepted as normal. The frequency varies with age, decreasing progressively from the child who has 110-120/min, to the normal adult with 80/min. In women the pulse rate is slightly higher than in men.

They also modify the frequency in the physiological state, rest and exercise. During exercise, it increases according to its intensity; Once this activity is completed, it decreases after a few minutes and returns to normal after an hour. Emotions also alter the pulse rate, making it faster.

Pulse rate:

In the normal individual, the pulsations follow each other rhythmically at equal distances from each other. The rate and rhythm of the radial pulse should be correlated with the rate and rhythm detected by precordial auscultation.

Respiratory sinus arrhythmia: when we count the pulse in several quarters of a minute we find that in some cases the number of pulsations varies, counting 16 in one quarter and 18 in others, and we also notice that these variations are accentuated with respiratory movements. For this reason it has been given the name respiratory sinus arrhythmia.

Pulse deficit: Pulse deficit can be detected by simultaneously palpating the peripheral pulse and auscultating the precordial pulse. There is a pulse deficit if the peripheral pulse rate is lower than that of the precordial pulse. Pulse deficits indicate that myocardial contractions are not strong enough to perfuse the extremities.

This condition can be observed in cardiac dysrhythmias, such as atrial fibrillation, atrial tachycardias or premature ectopic depolarizations.

Pulse hardness, amplitude and contour

Radial pulse hardness:

As mentioned at the beginning, the normal pulse offers resistance when trying to obliterate it with finger pressure, which is generally achieved.

Changes in pulse hardness:

Hard pulse: when the emptying of the radial artery is greatly difficult when compressing with the fingers. It is usually found in high blood pressure and arteriosclerosis.

Soft pulse: when when compressing the artery with the fingers, it depresses and empties easily. It is found in cases of shock and after hemorrhages.

Pulse amplitude and contour:

Pulse contour and amplitude indicate pressure and volume relationships across the vessel and are difficult to assess by palpation. However, the contour and amplitude can be easily evaluated by examining the pressure waves obtained by monitoring intra-arterial pressure.

Normal amplitude is represented by pulse pressure or differential pressure (the difference between systolic and diastolic pressure) of approximately 30-40 mm Hg.

This pulse registers 3+ on a scale of 0-4+.

The normal contour is characterized by a smooth upward inscription. The dicrotic notch represents the closure of the aortic valve (Fig. 12.3).

Auscultation

Auscultation of arterial pulses should not reveal sounds.

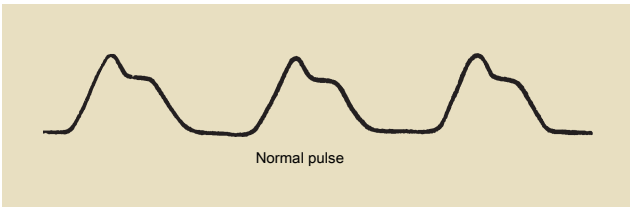
Auscultation of the abdominal aorta and the bilateral iliacofemoral sector is performed by applying the bell of the stethoscope over the umbilical region and in both iliac fossae up to the inguinal regions.

Rumors. A rumor is the vascular equivalent of a murmur and can be heard as a blowing sound caused by restriction of blood flow through the vessels.

STEPS FOR EXAMINATION OF THE PERIPHERAL ARTERIAL SYSTEM

1. Palpate the temporal arteries simultaneously.
2. Palpate each carotid pulse separately.

Fig. 12.3 Normal pulse.



Remember to avoid the carotid sinus, palpating much below the level of the upper border of the thyroid cartilage and that palpation should be gentle and shallow.

3. Locate the radial pulse.

Place the pads of your first three fingers over the pulse point of the radial artery, on the inner surface of the wrist over the radius. The thumb should not be used, because it has its own pulse.

4. Compare synchronism and amplitude:

a) Identify the presence, amplitude and synchronism of both radial pulses at the same time. If both are present, synchronous and normal, it is not essential to palpate the rest of the pulses in the upper limbs, at least to detect their presence and synchronism.

b) Compare the synchronism of the radial pulse with the femoral pulse on each side.

c) Now simultaneously identify the presence, amplitude and synchronism of both pedal pulses.

With the subject lying down, standing facing his feet and placing your hands as if you were going to clamp both feet, so that the thumbs face the plantar region and the rest of the fingers in the dorsal region of the foot, palpate with the index and middle fingers the indicated location of the arteries.

you asked. Simultaneous palpation can be performed using the left hand to palpate the right pedis and vice versa, but it can also be performed by crossing the arms in the midline, to palpate the right pedis with the right hand and vice versa.

If both pedal pulses are present, synchronous and have the same amplitude, it is assumed that the rest of the arterial pulses of the lower limbs, located higher up, are normal and in practice it is not necessary to routinely palpate them.

However, get used to always palpating all the arterial pulses of all four limbs, until you completely acquire this skill.

5. Evaluate the characters of the arterial wall and radial pulse wave.

Press firmly against the artery and slowly increase pressure until a pulse is palpable.

Too strong pressure obliterates the pulse. Thus, assess the ease or resistance to palpation and the character of the arterial wall, the hardness, amplitude and contour of the radial pulse.

6. Count the radial pulse rate.

Use a watch with secondary and count the rate of the radial pulse in one full minute and compare it with that of the apical pulse by auscultation, which should be the same.

If the pulse is regular, subsequent evaluations are usually made by counting the rate in 30 s and multiplying by two or counting it in 15 s and multiplying by four; but if the frequency is irregular, always count in a full minute, or in several minutes to obtain an average.

7. Assess radial pulse rhythm.

Always do this by first palpating the radial pulse alone and then while simultaneously listening for the tip beat.

The radial pulse should have the same rhythm as the apical pulse.

If any arrhythmia is detected: identify an arterial pulse deficit.

A pulse deficit exists if the apical frequency is greater than the radial frequency. This can occur in arrhythmias such as extrasystoles or atrial fibrillation and in heart failure, when the contraction is too weak to spread to the periphery.

8. Perform pulse auscultation:

a) Auscultate the temporal arteries, if you found any thrill on palpation.

b) Auscultate the carotids in search of murmurs, especially if you detected any murmur or thrill upon palpation.

c) Auscultate the abdomen to detect murmurs; in the umbilical region, for the abdominal aorta; on both sides, for the renal arteries; and in both iliac fossae, for the iliacofemoral sector of each side.

9. Record your findings:

a) If the entire examination is normal, describe at least that they are present, of good amplitude and rhythm, that they are synchronous (except for both carotid pulses, which were examined separately) and that no arterial murmurs are heard.

b) Don't forget to record the radial pulse rate in one minute (e. g. PR: 80/min).

c) The examination of the peripheral arterial system is completed with the measurement of blood pressure, which we will study below, and which must be recorded after the radial pulse frequency.

STUDY OF BLOOD TENSION

Concept. Following Gallavardin's classic criterion we can say that it is a force created by the heart, maintained by arterial elasticity and regulated by peripheral resistance.

Blood circulates in each of the two circuits, major and minor, with a gradually decreasing mean pressure from the ventricle to the atrium, where

reaches the lowest values. This is a functional property of blood circulation that is essential to be able to fulfill the purpose of the circulatory system, that is, to deliver blood containing oxygen and nutrients to all tissues and collect carbon dioxide and waste products from them.

Determining the pressures in the different sectors (arterial, capillary and venous) of the two circuits is extremely useful in some circumstances. In the larger circulation it can be done easily with clinical methods;
On the other hand, it is necessary to resort to the catheterization technique to measure the pressure in the minor circulation, for which reason it is carried out only in special cases.

In all blood pressure we must determine the maximum, which corresponds to the ventricular systole (maximum or systolic pressure), and the minimum, which is nothing more than the pressure that remains after the previous one has vanished (diastolic pressure).

The maximum and minimum pressure maintain a homogeneous (although variable) correspondence, within certain limits.

In the normal adult over 18 years of age, the maximum pressure is around 110-130 mm Hg and the minimum is 60-80 mm Hg.

In children the pressure is lower, much more so in infants, but its figures and methods to obtain it will be studied in Pediatrics.

Production mechanism

Numerous factors influence and maintain the body's blood pressure, a pressure that we must consider as a biological constant in the clinical sense, since it tends to always regularize itself in a figure, adapting its increases and decreases to the needs that may arise. These factors can be related to each other and condition small physiological oscillations in pressure. Let's see the most important ones below.

contractile capacity of the heart

The main energy capable of producing blood pressure is the contraction of the cardiac ventricles. We can evaluate this energy by studying cardiac output, heart work, minute volume or stroke volume.

Logically, when contractile energy increases, blood pressure increases; When there is weakness of the myocardium itself and the contraction does not occur normally, the pressure decreases.

Arterial elasticity

It is an important property of the arteries, essential to maintain a certain minimum pressure.

da, constant (more or less), which is the diastolic pressure.
Furthermore, because arterial elasticity exists, the blood flow that should be interrupted, since it rhythmically leaves the heart, becomes continuous. Thanks to this property, the blood can progress and maintain its pressure. When the maximum tension reaches, the arteries dilate and immediately return to their previous state, through the action of the muscular and elastic layers.

From the semiogenic point of view it has special value, since there are diseases where arterial elasticity is lost and there is a tendency to increase blood pressure, such as arteriosclerosis.

Peripheral circulatory resistance

The pressure leaving the left ventricle decreases as circulation progresses through the arteries until it reaches the right atrium. Peripheral circulation offers resistance to the passage of blood that is an essential factor in maintaining pressure. This resistance is one of the most important elements, since when there is vasoconstriction, the pressure rises; and when there is a drop in peripheral vascular tone, a drop in blood pressure occurs.

Peripheral resistance is due to three factors: the resistance of the arterial walls themselves, the so-called "friction" on the walls and the decrease in the caliber of the vessels.

Volemia

The amount of blood circulating in the circulatory system is another essential factor in maintaining blood pressure. If, for example, volume is decreased, blood pressure tends to fall. This is what happens after sudden losses of blood, plasma, fluids, etc. On the contrary, the increase in the amount of circulating blood determines an increase in blood pressure as occurs in polycythemia.

Blood viscosity

When viscosity increases, as happens in polyglobulias, blood pressure tends to rise and. On the contrary, in cases of anemia of a certain intensity, in which density or viscosity decreases, blood pressure suffers a decrease.

Among the extracardiac factors that influence and maintain the body's pressure, we have: renal, nervous and humoral factors.

Kidney factors

The relationship that exists between the kidney and arterial hypertension was described by Bright in 1827 and Volhard and collaborators have taken these relationships into account as a pathogenic factor in nephrogenic hypertension (pale hypertensives).

Since Goldblatt, experimental nephrogenic hypertension has been related to the pathogenesis of essential hypertension; Indeed, restricting renal circulation by clamping the renal artery causes hypertension, which is caused by increased vascular resistance.

As a consequence of renal circulatory disorders, a humoral pathophysiological complex (renin-angiotensin-aldosterone) would be activated, which produces an increase in peripheral vascular resistance, which causes arterial hypertension.

Renin is stored in the form of granules in the smooth muscle fibers of the walls of the afferent arterioles of the glomerulus, adjacent to the macula dense.

It is probably released linked to a protein that reacts with the substrate angiotensinogen, manufactured in the liver, to first produce the decapeptide angiotensin I, which is converted mainly into an octapeptide pressor: angiotensin II by the action of an enzyme (by hydrolysis of two amino acids when passing through the lung or other tissues).

This angiotensin II acts, in turn, directly on the smooth muscle, producing vasoconstriction, which increases vascular tone, and also stimulating the secretion of aldosterone in the adrenal cortex, a hormone that also acts on vascular tone and retains sodium and water. It will be easy to understand that when there is an increase in renin or aldosterone, or both at the same time, blood pressure will rise and vice versa.

Furthermore, different authors are searching for the link between kidney alterations and the pituitary-adrenal system.

Renal alterations would be valid for secondary nephrogenic hypertension.

In essential hypertension, other associated mechanisms should be considered: humoral, neurogenic, endrocrine, hydroelectrolyte alterations, etc.

Nervous factors

The nervous system regulates all the functions of our body; The regulation of blood pressure is carried out by higher centers, most located in the hypothalamus and others in the medulla, from which efferent impulses arise and, in turn, receive afferent fibers.

Vegetative nervous regulation acts through the sympathetic, producing acceleration of the heartbeat and through the vag0 system, slowing or inhibiting them. In the periphery, the sympathetic produces constriction of arterioles and capillaries, determining, by decreasing continent, an increase in blood pressure. The vagus, on the other hand, produces vasodilation.

The afferent systems can come from the skin, the carotid sinus, the brain centers themselves, such as rre, sometimes, with brain tumors and, finally, they can be stimulated by factors of the highest category within the nervous system: psychic factors. The fact that psychic influence develops changes in blood pressure, increasing or decreasing it, is now beyond doubt.

Chemical mediators. In relation to the vegetative system, there is the theory of chemical mediators that in the synapse between the vegetative nervous system and the tissues, act by releasing substances, which are sometimes of the sympathicotonic type, such as adrenaline and norepinephrine, and others of the parasympathicotonic type, such as acetylcholine. Furthermore, the overproduction of catecholamines (adrenaline and norepinephrine) by the adrenal medulla causes pheochromocytosis syndrome.

take.

Humoral factors

There are substances in the blood that, due to their chemical activity, can influence blood pressure, for example, CO and lactic acid.

The decrease in O tends to cause vasodilation of the arterioles with increased blood flow.

We have already pointed out the action of adrenaline and acetylcholine in relation to the vegetative system. Similar to acetylcholine is histamine, which is also capable of causing vasodilation and a drop in blood pressure.
Its increase in cases of anaphylactic shock would explain the existing arterial hypotension.

Other factors that influence blood pressure

Are:

internal secretions

The internal secretion glands exert a great influence on the maintenance of blood pressure.

In the pituitary-adrenal system, we consider that the anterior lobe of the pituitary gland has a common function with the adrenal cortex, as they produce hormones capable of raising blood pressure. There are numerous facts that demonstrate this: pharmacological, therapeutic, surgical, etc.

Aldosterone, as we pointed out, plays an undoubted role in blood pressure, and excess of this hormone leads to hypertension, which also appears in Cushing's syndrome of pituitary origin.

Constitutional and genetic factors

They have value when considering a tension figure.

The subject with an asthenic, long-line habit shows a tendency toward hypotension; while the brevilínea, picnic or plethoric, always shows a tendency to hypertension.
That is why it is important to assess against a moderate tension

Rarely elevated if it is an asthenic, longilinear subject, or if it is a brevilineal subject.

Dietary, social and toxic factors

The lifestyle you lead, your diet (excess), the use of alcohol, tobacco, coffee, etc., are factors to take into account.

There are toxins, some like lead and others like coffee, that tend to raise blood pressure. Alcohol by itself does not modify blood pressure.

Blood pressure examination

Blood pressure determination methods

Determination of blood pressure can be done by direct and indirect methods.

Direct method. Blood pressure can be measured directly, with invasive arterial catheters. In this method, the brachial or femoral artery is punctured with a cannula connected to a manometer or other pressure transducer systems. Although this method is of great value in some cases, it is impossible to use it in daily practice.

Indirect methods. They are for daily use in the clinic. They include the auscultatory, tactile or palpatory methods, the oscillometric method and the eclectic method, which combines the previous methods. The latter is the method that should always be used, as it is the safest and to which we will refer more widely.

Position of the examinee

During blood pressure measurement the subject can be standing, sitting or lying down.

An orthostatic or standing pressure should be assessed by first measuring the pressure while the person is lying down and then while sitting and standing. If the orthostatic value is significantly lower (> 30 mm Hg) it indicates orthostatic hypotension and may point to excessive volume depletion, prolonged immobility or neurological disease.

The extremity that will be used to take blood pressure should be positioned at a level equal to or lower than the heart to avoid a falsely low reading.

If the arm is used, the forearm should be in a relaxed position, for example resting on the table. Alternatively, the person's forearm should rest on your forearm.

When high blood pressure is suspected or detected, blood pressure measurement must meet important requirements to accurately achieve blood pressure measurement. The subject will rest 5 minutes before taking their blood pressure and must not have smoked or ingested caffeine for at least 30 minutes before taking it.

Korotkoff noises

During indirect blood pressure measurement, a stethoscope is used to listen for Korotkoff sounds.
These noises reflect changes in blood flow through the artery, as the cuff pressure of the sphygmomanometer decreases and the artery goes from a state of complete occlusion to maximum opening. Korotkoff sounds are generated by normal laminar blood flow being disrupted by cuff pressure, and the resulting turbulent flow creates vibrations in the vessel walls. There are five phases of different sounds.

Phase I. These are the first sounds heard as the pressure of the sphygmomanometer cuff decreases. The point at which the sounds are first heard represents the systolic blood pressure. The noises can be heard as clear taps that gradually increase in intensity for a short period of time, generated by rapid distention of the arterial wall as blood bursts into a previously collapsed artery. The intensity of the sound is related to the strength of blood flow.

Phase I. It can be heard as a soft murmur or delicate murmur. Murmurs represent turbulent blood flow and subsequent vascular wall vibration, created as blood flows from a relatively narrow artery, caused by inflation of the cuff, to a wider arterial lumen distal to the cuff. On many occasions, when the person has high systolic pressure, no noise will be heard in this phase. This silence is called auscultatory silence and can last 20-40 mm Hg. Failure to detect auscultatory silence represents a possible source of error in blood pressure measurement, because phase III noises can be misinterpreted as phase I noises.

Phase III. Phase III noises are intense, clear sounds, similar to those of phase I, but louder. Increased pitch and intensity distinguish phase I and II noise from phase III noise. In phase III blood flow occurs during systole, but cuff pressure remains high enough to collapse the vessel during diastole.

Phase IV. The sounds are different from previous noises;
These are off. The first sound of this phase occurs when the noise changes from intense to a dull sound and represents the first beat of diastolic pressure or first diastolic pressure.

Phase V. Occurs when the sound stops being heard, because normal arterial blood flow has been restored. He se-

Second beat or second diastolic pressure occurs when the muffled sounds can no longer be heard.

Equipment used for indirect measurement

Stethoscope and Doppler transducer

Korotkoff sounds can be detected by listening at a site of the pulse, distal to the pressure collection cuff. The sounds can usually be heard by placing the diaphragm of the stethoscope at the site of the pulse. If the sounds are difficult to hear, use the bell (Korotkoff noises are low frequency). If you still cannot hear sounds, place a Doppler transducer over the artery.

Sphygmonanometer

The term sphygmomanometer refers to the pressure collection cuff, connecting tubes, an air pump and a pressure gauge.

Pressure cuff or cuff. Korotkoff noises are generated when the properties of arterial flow are altered when the pressure cuff is inflated. The cuff has a distensible air pocket covered with fabric. A rubber tube connects the air bag to a rubber bulb that acts as a manual air pump, used to inflate the cuff. Another rubber tube connects the cuff to the manometer and indicates the air pressure inside it. The entire cuff should be wrapped around the limb and secured with a hook.

chos or adhesive cloth.

For a reliable measurement, the cuff should be wide enough to cover the upper two-thirds of the arm or thigh and long enough to completely encompass the entire diameter of the extremity. For the average adult arm, a cuff 12-14 cm wide may be sufficient. For the arm of an obese adult, or when pressure is taken on the thigh, a cuff 18-20 cm wide is used. Small sleeves are also available for children. If the cuff is too small, an abnormally high blood pressure reading may result. Similarly, if the cuff is too long, blood pressure may be underestimated.

Pressure gauge. The pressure gauge is the instrument that displays the cuff pressure in millimeters of mercury (mm Hg), an indirect reading of the subject's pressure.

There are two types of pressure gauges: the anaeroid, which uses a needle to point to the numbers on a calibrated dial, and the mercury gauge, which uses the height of a column of mercury in a glass tube to indicate pressure. The mercury manometer is read by looking at the meniscus of the mercury column.

curio at eye level and reading the corresponding number. Mercury pressure gauges are safer, simply because they do not require calibration. Anaeroid gauges are calibrated with mercury gauges using a y*" connector between the two gauges.

Electronic measurement

Electronic equipment is available to monitor indirect blood pressure, including continuous, non-invasive. The pressure cuff is applied to the arm, but the connecting rubber tubes are attached to an electronic monitor and inflation equipment instead of the hand pump and pressure gauge.

Auscultatory method

To determine the tension, the cuff is placed on an arm or thigh and insufflated with the bulb until the pulse beat in the humeral or femoral artery disappears. Applying the stethoscope at the level of the flexure region of the elbow or the popliteal region, it is auscultated while the air is slowly let out through a minimal opening of the valve located at the level of the pear. The moment in which the heartbeat is perceived marks the maximum or systolic tension, and when it disappears it corresponds to the minimum or diastolic tension.

Tactile or palpatory method

The cuff applied to the arm or leg is insufflated until the radial or pedal pulse disappears (supramaximal pressure). Then air is let out little by little until they reappear, a moment that indicates the systolic or maximum pressure. Then, by palpation of the humeral (below the inner edge of the biceps or its tendon) or the internal retromalleolar, in the same way as taking a pulse, the decompression of the cuff is continued, thus perceiving an increasingly intense and vibrant beat up to a maximum, from which the intensity of the beat decreases more or less abruptly. Record the point at which the first heartbeat is palpated as the systolic blood pressure. Diastolic pressure cannot be determined by palpation.

Oscillometric method

The first oscillometer of practical use was designed by Pachón and is the one that is still used, although slightly modified. It is composed of an aneroid manometer, which indicates the pressure inside the rubber cuff, and a manometric capsule, which indicates the amplitude of arterial oscillations. The measurement is made by decreasing the cuff pressure 5 mm Hg at a time, starting from a suprasystolic pressure to an intradiastolic pressure.

As the pressure decreases, the following phases are observed:

a) Supramaximal oscillations of small amplitude produced by the stump of the collapsed artery colliding with the rubber sleeve.
b) A sharp increase in the amplitude of the oscillations signaled by systolic pressure.
c) Progressive increase in the amplitude of the oscillations until reaching a maximum, followed by a decrease in the amplitude of the oscillations. This phase is called the phase of maximum oscillations.
d) A sudden decrease in the amplitude of the oscillations, which indicates the diastolic pressure.
e) Terminal oscillations of low amplitude that decrease progressively, called infra-minimal oscillations.

The data obtained with the use of the oscillometer:
systolic pressure, mean pressure, diastolic pressure and oscillometric index, can be obtained in any of the extremities or in a segment of them, and are very useful for the study of peripheral circulation.

Eclectic method for indirect blood pressure measurement

It is safer and more complete than the auscultatory and palpatory methods separately, because it combines the use of both. First, palpation is used to detect systolic pressure, thus avoiding falling into auscultatory silence and mistakenly taking as such the first beat of phase III of Korotkoff sounds, instead of the first of phase I. Auscultation is then used to corroborate systolic pressure and detect diastolic pressure.

Steps for taking blood pressure 1. Apply
the blood pressure cuff.

Take care to never apply the cuff to an extremity that has an implanted access connection to the hemodialysis equipment, such as an arteriovenous shunt or fistula. Repeated occlusion of the connection can contribute to clotting and limit its useful life.

Arm. Wrap the completely deflated cuff snugly and gently around your bare arm. The lower edge of the cuff should be approximately 2-3 cm above the antecubital space or elbow flexure (closer in children), and the center of the air pocket should be directly above the brachial or brachial artery. Improper cuff placement may result in an erroneous blood pressure reading.

Leg. Wrap the cuff around your unclothed thigh, with the bottom edge 2-3 cm above your knee.

she. The rest of the pressure measurement process is similar to that of the arm except that the artery to be palpated is the posterior tibial or popliteal artery and that the Korotkoff sounds must be heard over the popliteal artery, with the subject in a prone position, just as these pulses are palpated. Systolic pressure has values 10-20 mm higher in the legs than in the arms.

2. Estimate systolic pressure by palpation:

a) Palpate the radial artery with the fingertips of your non-dominant hand.
b) Inflate the cuff while simultaneously palpating the artery. To inflate the cuff, hold the pump or bulb in your dominant hand; Close its metal valve, turning it clockwise with your thumb and index finger, and then compress the pear several times.

c) Note the point on the manometer where the pulsation of the radial artery is no longer palpable. This allows a rough estimate of systolic pressure.

d) Completely deflate the cuff by turning the pump valve counterclockwise.

This part of the method foresees errors that could occur with the auscultatory-only method, due to not inflating the cuff enough and falling directly into auscultatory silence.

These steps can be skipped only if you are familiar with the examinee's usual blood pressure.

3. Auscultate blood pressure:

a) First find the brachial artery by palpation and place the diaphragm or bell of the stethoscope at this site.

b) Close the valve on the air pump again by turning it clockwise with the thumb and index finger of your dominant hand and inflate the cuff again, pumping the bulb, until the manometer reading is 20 mm Hg above the estimated systolic value, obtained by palpation.

c) Slowly deflate the cuff while auscultating the brachial artery. Deflate it at a rate of 2-3 mm Hg per second by turning the air valve on the pump counterclockwise. Rapid deflation usually causes erroneous readings.

d) As the cuff deflates, note the pressure gauge reading when you hear the first Korotkoff noise. This is the systolic pressure. Read the first diastolic pressure at the point where the sounds stop. Read the second diastolic pressure on

the point at which the sound disappears completely. Completely deflate the cuff and remove it unless a second measurement is needed.

Occasionally, you may deflate the cuff too quickly or there may be another reason to question the safety of the blood pressure reading. In such cases, you can repeat the blood pressure measurement. Wait one minute before reinflating, allowing blood flow to return to normal.

Read the pressure gauge at eye level to avoid error.

4. Repeat the procedure on the opposite limb (initial examination only). Check the blood pressure in the other arm and note if there is any difference. Take subsequent blood pressure readings in the arm with the highest pressure.

5. Record your findings.

The American Heart Association has recommended for many years that all three blood pressure readings should be recorded: systolic, first diastolic, and second diastolic. The record would appear as follows: 130/82/75. However, this type of record is only maintained for investigative work.

Despite these recommendations, many health professionals record only systolic and first diastolic, or second diastolic, pressure, such as 110/70.
In Cuba and in most countries in the world, the appearance of the first Korotkoff sound (phase I) is used to determine the systolic or maximum BP, and for the diastolic or minimum BP, their disappearance (phase V), according to the National Program for the Prevention, Diagnosis, Evaluation and Control of Arterial Hypertension, of March 1998, which follows the recommendations of the Sixth Report of the Joint National Committee for Prevention, Detection, Evaluation and Treatment of Arterial Hypertension (JNC-VI), published by the National Institute of Health of the United States in November 1997.

The limb used and the position of the subject should also be recorded so that later comparisons can be made. For example: "BP MSD" sitting: 120/80 indicates the blood pressure taken in the right upper limb, with the person sitting.
Don't forget to take and record the blood pressure in all four limbs the first time.

NORMAL BLOOD PRESSURE

Normal blood pressure figures vary according to different authors; We adopt the arterial hypertension criteria of the World Health Organization Health explained above, with the modifications suggested in our country by the National Hypertension Commission, to determine the normal figures in adults, from 18 years of age.

For adults 18 years or older

Category	Systolic (mm Hg)	Diastolic (mm Hg)
Optimal	Less than 120 and	less than 80 less
Normal	Less than 130 and 130-	than 85 85-89
Normal high	139	

Physiological variations

By continuously measuring blood pressure for 24 hours, it has been possible to establish its circadian rhythm, that is, the variations it normally has during the day. The lowest figure corresponds to deep sleep at 3:00 a.m. in the morning, after that moment it begins to rise and reaches its highest level between 11:00 a.m. and 12:00 a.m. It remains until 6:00 p.m. in which it begins to descend again, to reach its lowest level at 3: 00-4:00 a.m. In most people, blood pressure decreases by 10 to 20% during the night.

Among other factors that determine physiological variations in blood pressure we have age; The tension increases proportionally with the years.
The climate also affects blood pressure: it is estimated that in hot climates, blood pressure numbers are generally lower. Blood pressure is undoubtedly influenced by lifestyle. Thus, the hectic life led in cities influences a greater range of blood pressure figures, if we compare them with the figures recorded in places where life is calmer.

Individual variations

To these general physiological variations we can add some that we would classify as individual variations, and they are those that depend on rest or activity. Blood pressure rises with physical exercise and falls with rest, in such a way that it falls with rest, especially prolonged rest, that it can reach the lower limits of physiological blood pressure, limits that we call basal values. That is why physical effort and psychological tension constitute an individual variation factor. Both factors increase tension. Digestion also influences blood pressure; During the digestive period, blood pressure increases by 10-20 mm Hg. Sleep as a physiological influence factor acts on tension; during sleep the pressure is lower.

Regional variations

Finally, we are going to refer to regional variations. Blood pressure is the same in equal segments of the limbs; Thus it must be the same in both arms, in both legs, in both thighs. However, there may be differences from one arm to the other.

These normal differences between one and the other should not exceed 10 mm Hg. Any difference in blood pressure of more than 10 mm Hg should be considered pathological. There are pressure differences between the upper and lower limbs; Generally, in the lower limbs there is a slightly higher pressure, around 10-15 mm Hg. When it is greater or when it is reversed, it is a pathological case. Differences in the same member also exist but are of small importance. Generally the distal portion of the limb has a lower pressure than the proximal one; but the difference should not exceed 10 mm Hg; when it is older it is abnormal.

Pathological variations

Pathological variations in blood pressure are arterial hypotension and arterial hypertension and will be studied in Section II.

Arterial hypotension is studied in Chapter 47, which deals with alterations of the perivascular system.

ferric and high blood pressure in Chapter 45, when explaining the high blood pressure syndrome.

PERIPHERAL VENOUS SYSTEM

The examination of the peripheral venous system includes the examination of the jugular venous pulse, the existence or not of jugular engorgement and hepatojugular reflux, venous dilations, especially in the lower limbs (varicose veins, microvarices and vixens) and of collateral circulation, as well as, in specific cases, the examination of the central venous pressure.

As all of the above are alterations of normality or are explored in a sick individual, they will be described in this same volume, but in Section II, in the chapters corresponding to the cardiovascular system.

Suffice it to say for the moment that the recording of an examination of the peripheral venous system in a normal subject should reflect similar to the following:

Venosoperipheral system. No varicose veins or microvaricose in lower limbs; no collateral circulation, jugular engorgement or hepatojugular reflux. The jugular pulse is not observed. Central venous pressure: not explored.

GENERAL PHYSICAL EXAMINATION OF THE NERVOUS SYSTEM.

STATE OF MIND. FACIES. ATTITUDE AND GAIT

13

Introduction

The physical examination of the nervous system requires a different methodology than other systems, and although it uses the same procedures (inspection, palpation, percussion and auscultation), it mixes and combines them in a peculiar way, which we will describe as we study this type of examination in each of its parts. The physical examination is carried out with an orderly anatomical-functional criterion and not by examination methods or procedures.

Example: in the nervous system, facies, attitude and gait are studied by inspection; motility by inspection, palpation and combined maneuvers; trophism by inspection and palpation;
reflectivity by inspection, palpation, percussion and combined maneuvers; etc. As can be seen, the examination method is different from that used in the circulatory or respiratory system in which they were studied: all signs detectable by inspection, all those obtained by palpation, all signs detectable by percussion, and those that can be collected by auscultation.

Before entering into the evaluation of the nervous system in the general physical examination of the person (facies, attitude and gait), it is necessary to address different general aspects that must be taken into account in the comprehensive examination of the subject from the beginning of the interview, even before the general physical examination and which constitute the first focus of the particular examination, and the recording, of the nervous system.

STATE OF MIND

The evaluation of the mental state is done from the first contact with the subject, with the observation of behavior and the evaluation of the answers to the questions you asked during the interview or in your communication with the person, from the beginning of the physical examination. You just have to keep in mind that when you ask, for example, the name, age, address, or order the subject to choose a certain position, you are also evaluating the subject's mental state. If the communication and responses are adequate, there is no need to repeat each of the questions or mental status examination techniques when performing the particular examination of the nervous system. But if in these first moments you de-

detects any alteration that may interfere with the proper development of the physical examination, it is advisable to first evaluate the mental state, and even the entire examination of the nervous system, in order to know how to approach the rest of the physical examination and its limitations in terms of the techniques that require adequate communication with the subject.

Although it is true that the mental evaluation techniques are carried out in detail in subjects who are suspected or already have neurological alterations, it is necessary to know them from now on, to be able to conclude that the mental status examination is normal, to be able to carry it to the end, if any abnormality is detected and to be able to adequately evaluate the mental state of individuals with psychoneurophysiological alterations, but otherwise organically healthy, as happens in the elderly.

The mental status examination is the process of evaluating the functions that are controlled by the cerebral cortex, such as the ability to think, understand and interact with the environment.

Signs indicative of mental status can be revealed by people's appearance and behavior, as well as by responses to certain questions or instructions.

One way to demonstrate such signs is to perform the mental status examination in an orderly manner that includes the following:

- Level of consciousness.
- Orientation (in time, space and person).
- Communication skills.
- Memory.
-Language.

Level of consciousness

Consciousness refers to the level of wakefulness and responsiveness and is regulated by the ascending reticular activating system (RAAS) in the brain stem (see "Comatose Syndrome in Chapter 62, Volume 2).

It must be clear that in the strict sense, consciousness is not the same as orientation. A person may be conscious, but disoriented in time and space.

The level of consciousness is evaluated by observing the person's development, the ability to speak and follow verbal commands, and motor skills.

Primarily, it is classified and described in relation to the intensity of the sensory stimuli required to wake a person.

Terms that can be used to describe the level of consciousness are:

- Alert.
-Conscious, alert or fully awake.
- Obtunded.
- Stuporous.
-Comatose.

Although these terms are standardized and represent specific observations, some subjects cannot fit exactly into one of these categories. In these cases, the level of consciousness can be documented with a detailed description of the type of stimulus that was applied to make it react and its verbal or motor responses.

Alert or vigilant. It is the highest level of consciousness, characterized by the ability to respond to all types of sensory stimuli of minimal intensity, when external conditions require the individual to react as quickly as possible, and it is even manifested in electroencephalographic modifications. This level, higher than that of normal or waking consciousness, can even be pathological.

Fully awake, conscious or alert. Level of consciousness in which the individual is completely awake and oriented to time, place and person. Additionally, the person is able to respond to verbal commands.
However, we insist, an individual can be completely conscious, but remain disoriented.

Obtunded The subject appears drowsy or asleep most of the time, but is capable of spontaneous movements. It is possible to wake him up, but usually a gentle pat or additional shake is required to get him to say his name. A clouded individual tends to fall back asleep easily and may be disoriented.

Stuporous The subject sleeps almost all the time and makes few spontaneous body movements. More vigorous stimuli such as shouting or shaking him are needed to wake him up. He is still capable of responding verbally to these stimuli, although he does so incorrectly and for a short time, but he is less able or unable to follow orders and, if he tries to do so, his responses are wrong. A stuporous subject rarely orients himself or wakes up completely, even when the examiner is testing his response to sensory stimulation.

Comatose. The subject is unconscious most of the time and shows no spontaneous motor activity. Strong noxious stimuli, such as pain, are needed to provoke a motor response, which is usually a directed attempt to remove the stimulus. Verbal responses are limited to some monosyllables or absent. If no reflex response occurs, the individual is in a deep coma.

Exploration of the level of consciousness

The techniques for exploring the level of consciousness are based on the Glascow Coma Scale (see "Comatose Syndrome", in Chapter 62), which is a rapid, objective and reproducible test, where three parameters of the level of consciousness are evaluated: eye opening, best motor response and verbal response.

I. Determine the stimuli necessary to wake the patient

1. If the subject is completely awake, no further tests are necessary.
2. If the subject seems asleep or unconscious, try to wake him up by progressively applying increasingly more intense stimuli: calling him by name, touching him, shaking him gently, applying painful stimuli.
   Avoid unnecessary application of painful stimuli by first trying to awaken him with verbal stimuli.
   Certain methods of applying painful stimuli, such as vigorously rubbing the knuckles on the breastbone or poking the skin, should be avoided. Not only are they unnecessarily cruel, but they also cause major tissue damage and bruising.
3. If it is necessary to subject the subject to painful stimuli, these should be removed as soon as the response appears. The following methods can be used:

   a) Apply pressure to the trapezius muscle by grasping the belly of the muscle between your thumb and index finger and squeezing.

   b) With the same fingers, squeeze the Achilles tendon.

   c) Apply pressure to the nail bed, squeezing it between your index fingers and thumb. Some examiners prefer to place a pen or pencil on the nail plate and press the nail bed between the pen and index finger with their thumb.

   d) If there is no obvious response, some clinicians prefer to pull the short hair at the beginning of the temple, in front of the ear, which is usually very painful.

   e) Only if it is strongly suspected that the subject is feigning unconsciousness or that he is having an attack of conversion hysteria, breathing can be prevented for about a minute, covering the nose with one hand and the mouth with the other, which is very dangerous if the individual is truly unconscious and is performed more as a therapeutic method, when one is convinced of the absence of neurological alterations, than as a diagnostic method of the state of consciousness.

It is advisable to point out that the need to apply must be explained in advance to the family members present. describe these maneuvers, so that they are not misinterpreted as attacks that violate ethics or iatrogenesis.

II. Determine motor responses to verbal and painful stimuli.

A decreased level of consciousness is associated with a decreased ability to respond appropriately.
ently to the stimuli.

1. Able to obey verbal commands: the individual can move his limbs when asked, or perform some actions such as clenching and releasing his fingers.

   If the person is unable to release their fingers, the initial grasp may represent a reflex movement, rather than a voluntary motor response to the verbal command.

2. Able to locate or make proposed movements: the subject can try, or carry out, locate and stop painful stimuli.

   Such attempts, whether successful or not, should be classified as fully proposed moves.
3. Semi-proposed response: the subject grimaces or briefly flexes the limbs, in response to a painful stimulus, but does not attempt to withdraw the stimulus.

4. Flexor or decorticated postural response: flexion and adduction of the upper extremities with extension, internal rotation and plantar flexion of the lower extremities. It can occur in response to painful stimuli or spontaneously. It is associated with brain damage to the internal capsule or pyramidal tract above the brain stem.

5. Extensor or decerebrate posture: rigid extension and adduction of one or both arms and extension of the legs.

   It can also occur spontaneously or in response to painful stimuli. It is associated with little or no nerve activity above the brainstem and is a bleaker prognostic sign than decorticate posturing.

6. Flaccid response: no motor response to painful stimuli and a lax, weak appearance of the extremities.

ORIENTATION

Orientation refers to the subject's ability to understand, think, feel emotions, and appreciate sensory information about themselves and their surroundings.
At the highest level of orientation, people are able to react to sensory stimuli in a complete and expected way, with appropriate thoughts and actions.

It is evaluated by identifying the subject's level of orientation in relation to time, space and person.

The criteria for ruling out diagnoses of confusion, disorientation, and impairment of thought processes are partially based on the person's ability to make reliable statements about temporal events, their surrounding physical environment, and the people around them. However, be careful when interpreting criteria related to time, place, and person. Some subjects may respond in a seemingly inappropriate manner, when in fact they are oriented. For example, if you ask the day of the week and the subject does not know, do not conclude that he is disoriented (especially if he is hospitalized). Concluding that he is disoriented may be as wrong as concluding that a person who asks the date when he is going to write a letter, or a doctor to write a prescription, is confused.

On the other hand, a person may be oriented to time, person, and place, but have a relative deficit in orientation to self and others. This condition is associated with damage to the right cerebral hemisphere, particularly when there is left hemiplegia. The person ignores perceptions related to or originating from the right side of the body, which has important implications for self-care. Homonymous hemianopia, a visual disturbance characterized by blindness in the nasal half of one eye and the temporal half of the other, may accompany forgetfulness of one side.

Orientation exploration

I. Evaluate orientation in time, space and person

When orientation deteriorates, the person usually loses time orientation first, followed by place orientation, and then person orientation. However, exceptions to this pattern can be observed.

1. Determine the orientation in time.

Evaluate the answers to the following questions:

a) What is the date (day, month, year)?

b) What day of the week is it?

c) What time of day is it (morning, afternoon, night)?

d) What was the last meal you ate (breakfast, lunch, dinner)?

e) What season are we in?

f) What was the last holiday?

People in unfamiliar environments, without elements that indicate time, such as clocks, calendars, television, newspapers, can lose track of time. The last four questions on this list can have a more appropriate meaning for the evaluation of such people.

2. Determine the orientation in space (place).

Evaluate the answers to the following questions:

a) Where are you now?

b) What is the name of this building?

c) What is the name of this city?

d) What province is this?

Consider whether or not the person has been transferred multiple times (for example, whether they have been transferred to several hospitals, departments or units or wards). In such cases, the person may have difficulty naming the current location where they are or the previous one.

3. Determine the orientation of the subject on your person.

Evaluate the answers to the following questions:

a) What is your name?

b) Who was visiting here now?

c) Who is this? (indicate visitors or family members who are present).

d) What do you live on? What are you doing? What do you work on?

e) How old are you?

f) Where do you live?

g) What is the name of your wife (husband)?

Educate yourself in advance about identity data (name) and other personal data, including roles and lifestyle. Asking a person to name several members of the health team may be an inappropriate technique, especially if the subject has been in contact with many different people in one day, as occurs in emergency rooms, etc.

II. Assess for the presence of "one-sided abandonment"

This type of evaluation is indicated for subjects who appear to ignore sensory messages from the left side of the body.

1. Observe how the person walks. He often stumbles over things on the affected side, usually the left.

2. Observe the person's performance of daily activities. Ignoring one side when bathing, combing your hair, shaving, dressing or eating is a sign of abandoning one side."

3. Place some small common objects on a table in front of the person. Ask him to name them. If there is "one side abandonment", it fails to name the objects located on the left side.

4. Ask the person to read a full-width headline.

the newspaper page. The person with "one-sided abandonment" may skip words on the left side of the page.

5. Ask the person to draw a clock face or their self-portrait. The left side of the drawing is incomplete or missing, with "one side abandoned."

MEMORY

Memory is the ability to store thoughts and learned experiences and recall previously learned information.

Three types of memory are described: immediate or retention memory, recent or anterograde memory, and distant, evocative, or retrograde memory.

Memory scan

1. Examine immediate memory:

a) Ask the person to remember three numbers such as 7, 0, 4. A minute later, ask him or her to repeat the numbers.

If the person has difficulty with this operation, give them a simpler test of immediate memory or retention. For example:

b) Ask the person to take an object, such as a pencil, with their left hand and put it in their right hand.

If the person cannot retain all parts of the instruction, then simplify the test further.

ba:

c) Only ask him to hold the pencil with his left hand.

Tests to evaluate immediate memory indicate whether or not the person is capable of registering information in the memory cortex. The ability to successfully answer immediate memory tests indicates that it is intact and that the person understands its message. This rules out other problems such as receptive aphasia or apraxia (see below and Chapter 14) that could interfere with the person's ability to make appropriate responses.

2. Examine recent, anterograde, or fixation memory.

Recent memory tests indicate whether or not a person has the ability to remember new information a short time after it is presented.
Ask the person what they had for breakfast or similar questions about events that have recently occurred, such as if someone came to visit them that day.
The person may register immediate information (such as that expressed by repeating phrases or numbers), but forgets the new information within minutes and does not remember or confuses recent events. However, a person with recent memory loss may have clear memories of temporally distant events.

Recent memory loss is also called anterograde amnesia.

3. Examine distant, retrograde, or recall memory.

Ask the subject general questions about his or her distant past, such as year of birth, types of operations he or she has had, or where he or she grew up. Check your answers with family members or health history.

Ask general questions about the distant past that involve general knowledge. For example, ask the person to say the name of the Cuban dictator overthrown by the Revolution or what date the Cuban Revolution triumphed.

Keep in mind that answers to general questions may be inadequate if the person understands that the object of the question is irrelevant to his or her interest, or if the educational or cultural level is such that the subject does not have the information necessary for a correct answer.

Retrograde amnesia is characterized by remembering only events from the very distant past, such as childhood.

4. Look for the existence of collusion.

Confabulation is the attempt to compensate for memory loss using fictitious information. In other words, the person can fabricate answers to questions and may even admit to this practice.

LANGUAGE

Definition of terms

Speech: articulation of the word. The formation of words through the use of special neuromuscular structures.

Dysarthria: the inability to speak properly, due to a neuromuscular deterioration of the tongue, lips, oropharynx, etc. , which allow the articulation of speech.

Language: the brain's interpretation of messages or their conversion into them, through listening, reading, speaking or writing.

Aphasia: loss of language ability. It can be of various types; The most important are: receptive or sensory aphasia, when the ability to interpret symbols and words is lost; motor or expressive aphasia, when the subject is not able to convert images, thoughts, into the symbols of oral language, words; and mixed or global aphasia, when the ability to understand and express oneself adequately is lost in both processes.

Praxia: ability to perform more or less automatic motor acts, which have a certain order, such as combing one's hair, and which require: the ability to identify

the objects to be used: the knowledge of what the object is used for, the ability to decide its use and the orderly execution of the action; everything, quickly and reflexively (see the examination of praxia, in Chapter 14 and a more detailed exposition in Volume 2).

Apraxia: inability to perform these motor actions, even when there is no evidence of paralysis, weakness or loss of coordination.

Anatomophysiological meaning

Speech and language functions are regulated by specific areas of the cerebral cortex. The left cerebral hemisphere is the dominant one, for language functions, in more than 90% of right-handed people. Therefore, strokes in the left hemisphere are commonly associated with aphasia, while those in the right hemisphere are less prone to it.

In left-handed people, the left hemisphere is dominant for language, only in 50-75% of people.

times.

Despite the concept of dominant hemisphere, it must be remembered that both hemispheres have language functions. The left hemisphere is responsible for propositional language, such as word order, selection, and combinations to form phrases or sentences. The right hemisphere is responsible for affective language, such as speech melody, emotional tone, and intonation.

Wernicke's area (Brodmann's No. 22) is located in the temporal lobe of the dominant hemisphere and processes word symbols, allowing comprehension and interpretation.

Broca's area (Brodmann's No. 44) is located in the frontal lobe of the dominant hemisphere and functions in aspects of propositional language and includes converting messages in the brain into words, phrases or sentences, in accordance with grammatical rules.

Another major speech and language area is located in the angular gyrus of the parietal lobe, which receives all sensory input and converts it into language.

At a minimum, you should pay attention to the person's way of speaking and their understanding of language.

A more rigorous evaluation should be initiated when there is obvious deterioration in speech and language functions.

Because language assessment can be embarrassing and frustrating for the examinee, assess communication and language processes only when necessary. Carefully record communication and language findings to avoid repeated testing.

When a subject cannot communicate, ask family members or friends about their previous language skills and ways of communicating, as well as their hearing and vision abilities. Also ask about their reading and writing abilities, because these abilities should also be evaluated. It is an erroneous conclusion to assume that a person has a brain disorder, because they cannot read, if the person has never learned, or because they do not understand, if they are a foreigner and do not master the language.

Language exploration

1. Assess understanding:

Remember that the evaluation of comprehension must have already been done from the beginning and that these exploratory techniques are only carried out in detail if any language alteration is suspected or evident.

First determine if there is any indication that the person has a hearing problem.

To properly assess understanding, when you ask each question or issue an order, be careful not to make gestures that denote its content or suggest the answer.

a) Determine if the person understands by asking simple open-ended questions, such as the person's name, age, or address.

Receptive aphasia (Wernicke's aphasia, sensory aphasia) is characterized by impaired comprehension

b) Next, ask closed-ended (yes-no) questions, including some that are not based on reality, such as asking if she has a flower on her head.

Receptive aphasias can be of varying degrees; In milder cases, the person may have difficulty just interpreting the name of certain objects.

c) Determine the ability to follow simple directions, asking the person to follow simple commands, such as pointing to the clock or window, or touching their nose.

The inability to follow orders is characteristic of apraxia, as well as receptive aphasia, because in the latter case, the individual does not understand the order given to him.

d) Name an object and ask the person to point to it; or show him an object, such as a pencil, and ask him to respond by moving his head, if it is a scissors, a shoe, a comb, a pencil.

As we will see below, a subject may not be able to say the name of an object (motor aphasia),

but you will recognize your name when someone says it, if you do not have receptive aphasia.

2. Evaluate oral expression:

a) Ability to speak fluently. Observe the use of complete sentences, or phrases, in the person's conversation. Also observe if there is any facial deviation, to rule out dysarthria.

Expressive aphasia (Broca's aphasia, motor aphasia, non-fluent aphasia) is characterized by impairment of speech abilities.

The conversation may be non-fluent or telegraphic, or there may be a pause in speaking. The person may speak like a child, using only nouns and verbs. For example, I hunger, urinate. The person may struggle to form words or may struggle to produce sounds. The telegraphic way of speaking is characterized by truncated messages. There may be preservation, which is the tendency to repeat words or sounds; for example, when, when, when.

b) Repetition capacity. Observe how easily the person repeats words and phrases when instructed to do so. Note whether or not there is word substitution with this operation (exercise). Repetition capacity is poor in expressive aphasias, because the person must struggle to form words. Repetition capacity is also poor in receptive aphasias, due to paraphasia errors; For example, replace " give" with "I stop using slang.

c) Ability to name. Point out specific objects and ask the person to name them.

The ability to name may be impaired, both in expressive and receptive aphasias.

you go.

3. Assess written expressive language:

a) Simple writing exercises. Ask the person to write their name and address.

Writing abilities are poor in expressive aphasias. There is a pause in writing, which parallels the ability to speak.

b) Complex writing exercises. Ask the person to write a short paragraph. For example, ask him to write about what he saw on TV or what he ate for lunch.

Writing abilities may also be poor in receptive aphasias, parallel with speaking abilities. The person may be able to write, but the writing will have no meaning.

4. Assess extraverbal language:

a) Note the appropriate use of gestures.

b) Observe the range of emotions demonstrated in general gestures, facial expression, and tone of voice.

VOICE.

c) Look for flat affect (not signs of emotions).

d) Observe affective lability (extreme fluctuations of feelings and emotions).

FACIES

Each of the two halves, right and left, of the face has complete symmetry in its muscular and nervous structure.

The muscles of the face are those that carry out the actions of mimicry, a way of expressing the emotional state or thought without the use of words, as well as giving emotional expression to the word itself.

This mimicry is part of the so-called extraverbal language, of no small importance in medicine. All the functions or influences of the central nervous system are integrated into the expression of the face, including the personality of the

The muscles of the face are innervated by cranial nerves, which, like the two halves of the face, are arranged in pairs. These cranial pairs come from peripheral motor neurons located in nuclei or neuronal groups along the brainstem (midbrain, pons and medulla oblongata), on the same side of the hemiface that they innervate (with the exception of the IV nerve).

Each motor nucleus of a cranial pair simultaneously receives pyramidal motor fibers from the cerebral cortex of the opposite side, coming from the lower part of the motor cortex (precentral gyrus or ascending frontal or prerolandic gyrus).

The motor nuclei, as the final common pathway of the other higher nervous centers, also receive extrapyramidal, cerebellar, etc. influences.

When we study muscle tone, we will see that this particular state of semi-tension that a muscle normally has at rest, and which in the case of the face gives it its normal appearance, also depends on the indicated structures, both the peripheral ones (cranial nerves, muscles) and the central ones (cerebral cortex, psyche, pyramidal, cerebellar and reticular pathways). Alterations in muscle tone cause alterations in facies.

ATTITUDE

The erect position is characteristic.

In the adult state, the man can adopt the lying, sitting or standing attitude. The newborn is inca-

peace of adopting another attitude than lying down. A sign of its satisfactory evolution is trying to acquire, as it grows in age, the standing attitude, which requires a long learning process.

The standing attitude is achieved thanks to an active neuromuscular mechanism. It is a static motor activity that requires the cooperation of a large number of reflexes, especially tonic ones.

This position contrary to the laws of gravity is achieved and maintained thanks to the tonic contraction of the muscles that have an antigravity function, mainly the extensors, and among them: the retractors or extensors of the neck; the masseter and temporal bones, responsible for keeping the lower jaw elevated; the supraspinatus; the extensor muscles of the back, and the extensors of the knee, leg, thigh and foot.

Proof of what we have pointed out before is the fact that a person who has these muscles relaxed, without tonic contraction (as occurs in an unconscious person), cannot maintain the standing attitude, but falls due to the action of the force of gravity, and adopts the lying position, indifferent, on the floor, bed or table, depending on where he is before losing consciousness.

When we study later the intimate mechanism of muscle tone, we will better understand what the contraction of a muscle is and we will see that it depends mainly on the passive stretching of the muscle itself, which creates a reflex contraction of the same (myotaic reflex), which is what originates and maintains the tone.

These myotatic reflexes have centers in the spinal cord. Higher centers act on these spinal centers whose function is to regulate (increase or slow down) the muscle tone itself, and which are normally all acting simultaneously, so that a tonic activity appropriate to each muscle results, in accordance with the purpose or function it is performing at each moment.

These spinal centers receive, in turn, sensory, tactile and pressure stimuli, produced by the contact of the sole of the foot with the ground (even through shoes); stimuli from the labyrinth, produced by changes in the position of the head in space; and stimuli created by visual and auditory perceptions that determine head movements.

A characteristic of tonic muscle contraction is the little expenditure of energy it implies, which allows maintaining an attitude for a long time and with little fatigue, as occurs with the contraction of the masseters that keep the jaw elevated and the mouth closed for most of the day.

It is assumed that this is because in this type of contraction only a limited number of motor units Each muscle contracts at all times, periodically alternating between contraction and rest, the different groups of fibers of the same muscle.

The standing attitude is unstable and requires constant variation in the intensity of the tonic contraction of the different muscle groups responsible for maintaining it, according to the oscillations of the body and the variations in the weight that each muscle group must support at all times.

From all of the above it follows that to maintain the standing attitude under normal conditions, it is required:

1. Integrity of consciousness.
2. Integrity of the locomotor system (bones, joints, muscles, tendons, ligaments, etc.).
3. Integrity of the nervous mechanisms responsible for maintaining muscle tone:

a) Of the peripheral, motor, sensory and mixed nerves.

b) Of the medullary centers of tone.

c) Of the higher centers that regulate the previous ones

beef.

MARCH

CONCEPT

Gait is defined as the normal or pathological way or style of walking. The march is the result of a series of coordinated acts of voluntary initiation that are then carried out automatically. It requires, therefore, a determinative element and an executive and coordinating cerebrospinal element.

PRODUCTION MECHANISM

Walking is a complex activity, the result of the integration of numerous physiological mechanisms among which the nervous system is one of the main ones but not the only one.

For the march to take place normally, it is required:

1. Mental state of wakefulness that allows making the decision to carry out the march, which is a volitional act.
2. Indemnity of the following nervous structures and functions:

a) Pyramidal route, way to execute the volitional act.

b) Cerebellum, coordinates all movements so that The march has adequate measure and synergy. Synergy is the correct association carried out in time of the various elemental movements that comprise walking. The adequate measurement of movements (eumetry) allows each of

They are carried out without exceeding or falling behind the point required for the purpose proposed. The cerebellum is also involved in the regulation of muscle tone.

c) Cerebellar pathways, both afferent and efferent.

d) Extrapyramidal system; It intervenes in muscle tone and the conditioned reflexes necessary for walking.

e) Vestibular nerve and pathways; fundamental in balance.

f) Spinal cord; In this segment the following are important:

You make gracilis (Goll) and cuneatus or cuneiform (Burdach); They conduct all proprioceptive information to the higher nervous centers.

Side drawstring; It contains the pyramidal or corticospinal tract.

Motoneuron of the anterior horn of the spinal cord; executor and final common pathway of motility, muscle tone and other impulses necessary for movements.

g) Peripheral nerves of the lower extremities;

They collect information from peripheral receptors and also carry the response from the motoneurons of the anterior horn to the osteomyoarticular system.

3. In addition, other structures not belonging to the nervous system are necessary, the alteration of which can affect gait:

a) The vision.

b) The inner ear.

c) The muscles of the lower limb, the pelvis and those related to the spine.

d) The bones and joints of the vertebral column, the pelvis and the lower limbs.

e) The circulatory status of the legs (arterial, venous and lymphatic, especially the first).

f) The general, physical and mental state of the subject.

EXPLORATION TECHNIQUES

During walking, not only the trunk but also the arms are in motion: the upper limbs move at the same time as the lower limbs, but In the opposite direction, the left arm goes backwards while the left foot goes forward.

These movements are intended to partially correct the displacement of the center of gravity, due to the movements of the legs and the twisting of the trunk.

As the pace of walking increases, the amplitude of the arm movements decreases; These flex to be able to oscillate more quickly, a circumstance that explains why any effort to cancel or counteract the movement of the arms results in hindering walking. The same will happen if they are exaggerated.

The subject is made to walk, lightly clothed, in a spacious, well-lit room with adequate temperature.

It is examined in profile, walking forward towards the observer and away from him.

We will pay attention to whether he looks ahead, as usual, at the ground (with his eyes on the movements of his feet and where he steps) or at a fixed point located at mid-height (eye crutches); if the march is fast and determined or if it is slow and clumsy, without natural grace and gallantry; if it follows a straight line or if it deviates: if it separates the lower limbs too much in search of a greater base of support or, on the contrary, if it brings them too close together; whether or not there is anteroposterior tilt of the pelvis and scapular compensation; if the arms swing normally (the right one advances when the left foot does, and vice versa), if they remain rigid and attached to the body or, on the contrary, if they separate in search of support points or move without any purpose, as in choreics.

In the profile examination we observe the play of the thighs on the trunk, the knees and ankles and the feet, specifying which part reaches the ground first (the heel or the toe) and the support points.

The test is completed by having him walk a certain number of steps forward and a number of steps backward, first with his eyes open and then closed, as well as stopping abruptly and retracing his steps at an agreed signal, a clap, for example, to see if he does so safely or staggeringly (see exam of the VIII pair, in Chapter 15).

14

PARTICULAR PHYSICAL EXAMINATION OF THE NERVOUS SYSTEM.

TAXIA, PRAXIA, MOTILITY, TONE AND TROPHISM, REFLECTIVITY, SENSITIVITY

TAXIA

CONCEPT

The term taxia is synonymous with coordination. It is defined as the combination of contractions of agonist, antagonist and synergistic muscles that aims to achieve harmonious, coordinated and measured voluntary movements. The most important center for the coordination of the acts in which the muscles intervene is the cerebellum.

The term ataxia indicates a pathological state of incoordination of motor acts, which occurs despite there being no alterations in motility or muscles.

PRODUCTION MECHANISM

When performing a movement, several groups of muscles intervene simultaneously, the action of which is diverse, but in which they all contribute to the same objective. These muscles are:

1. Those essential to carry out the movement and are called agonist muscles.

2. Those that, having an opposite action to the agonists, relax simultaneously during their contraction, so as not to oppose the movement that is carried out, are called antagonistic muscles.

3. Those that help the two previous groups and that is why they are called synergist muscles.

4. Those responsible for fixing the joints neighboring the region that moves, in order to allow the displacement of a part of said region, which is what constitutes the movement and which (due to this fixing action of the joints) are called fixing muscles.

From all of the above, it can be deduced that for a movement to be effective, that is, adequate for the purpose or purpose, it is necessary that there be a mechanism in the nervous system that coordinates the execution of the movements. The study of taxia is the study of this system; It is logical that a lesion of this coordinating mechanism clinically produces sometimes very marked motility disorders, without paralysis or paresis of the muscles.

The following are involved in the complex mechanism of coordination:

1. The cerebellum.
2. The cerebral cortex (pyramidal and extrapyramidal systems).
3. The pathways of conscious and unconscious proprioceptive sensitivity.
4. The labyrinth and the vestibular pathways.
5. The vision.
6. The multiple and complex nervous connections between these centers that include some nuclei of the brain stem, the most important of which is the red nucleus.

The primary function of the cerebellum is to provide synergy to actions and postures. The cerebellum coordinates and modulates muscle contraction constantly, so that our body can remain stable in a standing or sitting position and balance is maintained during locomotion or any other muscular activity. The cerebellum acts on each of the muscles that participate in these actions so that they contract at the precise moment and to the necessary degree, which can be called chronometry.

The cerebellum is located in the posterior cranial fossa, below the occipital lobes and covered by the tentorium cerebellum. It maintains connections with the central nervous system through the so-called cerebellar peduncles, which are three pairs: superior cerebellar peduncles, which end in the upper part of the brain stem and contain the efferent pathways of the cerebellum and some afferents; middle cerebellar peduncles, which emerge from the pons and inferior cerebellar peduncles (corpus restiform), which, forming the external and inferior part of the IV ventricle, enter the cerebellum. These last two cerebellar peduncles conduct the afferent pathways to the cerebellum.

The cerebellum has a central portion called the vermis and two more developed lateral portions in humans, which are the cerebellar hemispheres.

The lower anterior and central part is the flocculonodular lobe. It is the old part of the cerebellum from a phylogenetic point of view and the one that maintains the greatest relationships with the vestibular nuclei. The vermis intervenes in the coordination of the trunk, head and limbs; while the cerebellar hemispheres, the phylogenetically newest part (neocerebellum), are considered to be involved in the finest coordination typical of man, such as that of the hand and the muscles that intervene in the expression of language.

Almost all cerebellar afferents reach the cortex of this organ, which has multiple connections with the cerebellar nuclei, accumulations of gray matter.

located in the central portion and which are the nucleus of the roof, the emboliform, the globose and the dentate. All cerebellar output comes from these nuclei.

Cerebellar afferents. The main ones are: the spinocerebellar (spinocerebellar, direct and crossed fascicles), the vestibulocerebellar, the olivocerebellar and the pontocerebellar (fig. 14.1)

Most of the fibers that ascend through the spinocerebellar fascicles come from dorsal and lumbar spinal cord segments, which receive information from the trunk and legs. Fibers also enter from the spinal column. These afferents enter the cerebellum through the inferior cerebellar peduncles and some through the superior cerebellar peduncle (crossed spinocerebellar fasciculus) and end in the cortex of the cerebellar vermis, which connects mainly with the emboliform and globose nuclei.

The vestibulocerebellar pathways terminate in the cortex of the flocculonodular lobe, which connects primarily to the tectum nucleus. These fibers carry information to the cerebellum regarding the position of the body in space (especially the head).

The corticopontocerebellar pathway has two neurons: the first is located in the cerebral cortex (frontal, temporal, parietal and occipital lobes) and ends in the ipsilateral region (on the same side) of the pons, in the nuclei of the anterior region, which constitute the second neuron of this pathway. The axons of these neurons cross forming the arcuate fibers of the pons, which entering through the middle cerebellar peduncles end in the entire cerebellar cortex, except the flocculonodular cortex.

Cerebellar output. The efferent pathways of the cerebellum, related to the muscular coordinating function, start from the cerebellar nuclei, travel through the superior cerebellar peduncles and reach the motor nuclei of the cranial nerves and the motor neurons of the anterior horn of the spinal cord mainly via the vestibular nuclei and the red nucleus (fig.14.2).

Some fibers terminate in the nuclei of the substantia reticularis and in the vestibular nuclei; others descend from the anterior cord of the medulla to end in the anterior horns. The vestibulospinal fasciculus also descends from the vestibular nuclei, and there are connections with the nuclei of cranial nerves III, IV and VI through the medial longitudinal fasciculus. But most of the efferent fibers of the cerebellum cross when entering the brain stem, later reaching the superior colliculus, the nucleus of Darkshevich, the interstitial nucleus of Cajal and the red nucleus; A large part of them ascend until they end up in the thalamus and from there project to the cerebral cortex.

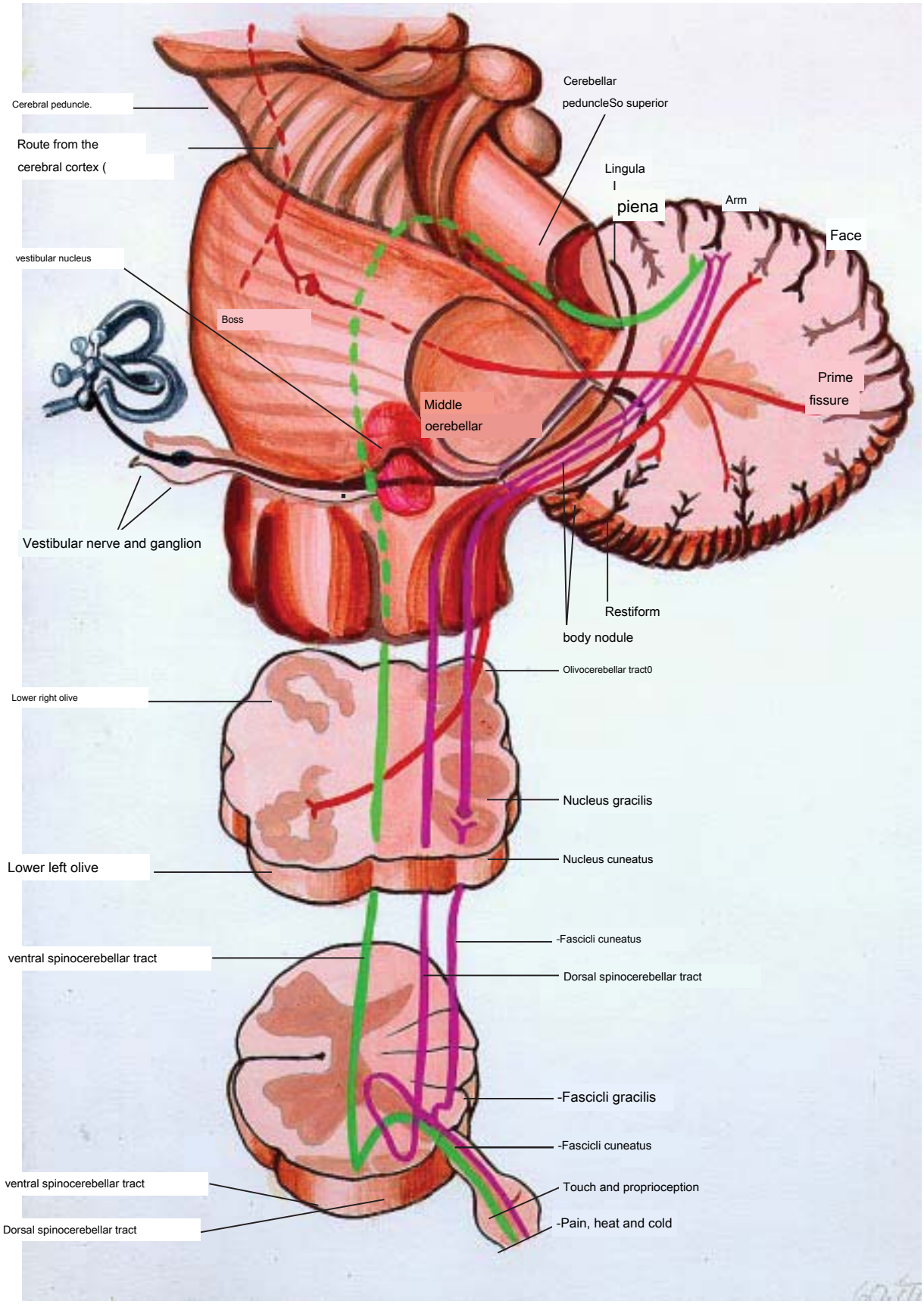


Fig. 14.1 Afferent connections of the cerebellum.

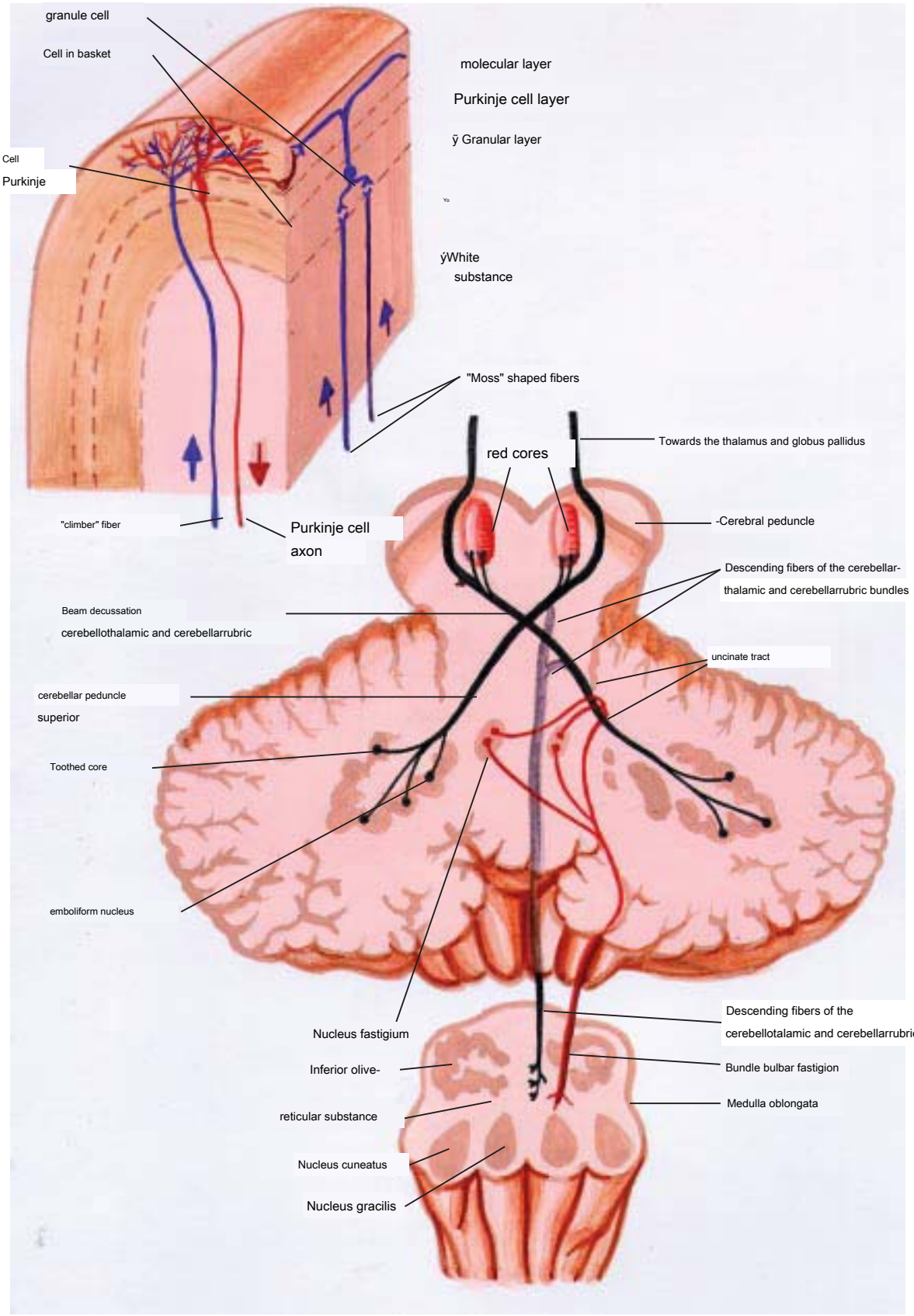


Fig. 14.2 Efferent connections of the cerebellum.

Fibers come from the red nucleus that establish connections with the reticular formation of the pons and the medulla oblongata, and with the anterior horns of the medulla (rubrobulbar and rubrospinal fascicles).

The cerebellum also influences certain visceromotor mechanisms such as pupillary dilation, bladder contraction and vasomotor responses.

Certain fibers even reach the cerebellum, coming from the gastrointestinal tract. The observation is cited that vomiting and vertigo disappear after ablation of the cerebellum or the flocculonodular lobe.

The presentation we have just given gives a vision of the multiple connections of the cerebellum, which allow this organ to exercise its function as a suprasegmental structure. Also through this knowledge we can understand the great importance that the cerebellum has in the coordination functions for which it has been called "direction ganglia of the proprioceptive system." All this delicate device needs, to reach its maturity, a certain amount of learning time; hence the so-called physiological ataxia in the first year of life.

EXPLORATION TECHNIQUES

The coordinating actions of the central nervous system can be static and dynamic (locomotor or kinetic).

Exploration of static coordination A.

Simple Romberg maneuver.

1. Order the subject to stand with his feet together, to maintain the military posture of “firm.” The doctor at your side will be attentive so that the individual does not lose balance and fall. Observe whether or not he experiences oscillations in this position.
2. Now instruct the subject to close their eyes. Observe then, if it maintains its balanced position or if, on the contrary, its body oscillates and has a tendency to fall (fig. 14.3 a).

In this case it is said to present Romberg's sign.

This sign can only be accepted as positive when there is a real loss of balance during the maneuver with the consequent separation of the feet to maintain it. Oscillations of the trunk, without loss of balance, should not be considered as Romberg's sign, since it is typical of many neurotic people.

B. Sensitized Romberg maneuver.

It is performed only if the simple Romberg maneuver is negative, that is, if it did not obtain a Romberg sign.

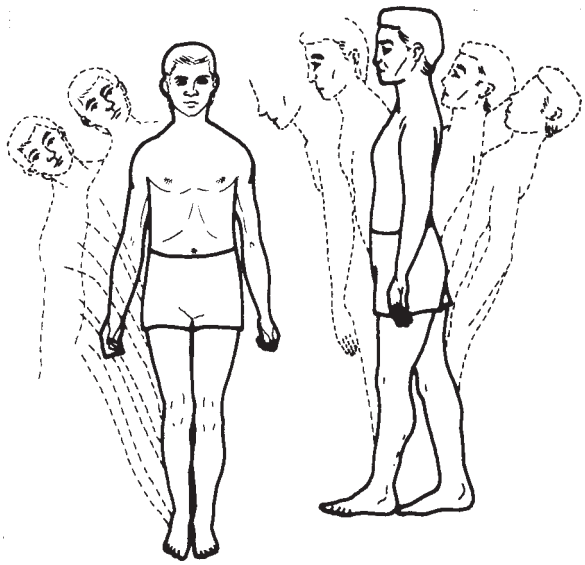


Fig. 14.3 Simple Romberg maneuver (a) and sensitized Romberg maneuver (b). The dashed images represent examples of Romberg' s sign.

1. Order the subject to stand with one foot in front of the other (fig. 14.3 b) and try to maintain balance. You can help yourself with your arms extended to the sides.
   It can also be done with one leg raised up to the knee of the other, forming a kind of number four.

2. Then, you are instructed to close your eyes and observe if your balance continues to be maintained or if your body oscillates and tends to fall. In these conditions it is more difficult, even normally, to maintain balance. Keep in mind that the elderly, and even subjects who are not elderly, find it difficult to maintain balance, especially with the 4" position, even with their eyes open. Do not insist on performing the maneuver in this last position in these cases, nor in those who are ill for another reason, in whom there is no suspicion or evidence of alterations in static coordination.

Exploring dynamic coordination

All the following tests are done first with eyes open and then with eyes closed, to avoid rectification through the sense of sight. Since the positions and actions to be performed in these maneuvers are sometimes difficult to understand, because it is the first time that the person performs them, in addition to explaining them, try to demonstrate their execution.

1. Finger-finger or finger-thumb test. It consists of touching the tip of the thumb with the tip of each of the other fingers successively and quickly, first with one hand and then with the other.

2. Index-index test. The person is asked to open their arms with their index fingers extended and then close them in front, ensuring that the tips of the index fingers touch. It is done several times with eyes open and then with eyes closed.
   If there is no alteration of the taxia, the person performs this with little or no difficulty; If there is ataxia, it will be done with insecurity, so that the indices do not conflict and one goes higher or lower, further forward or further back than the other.

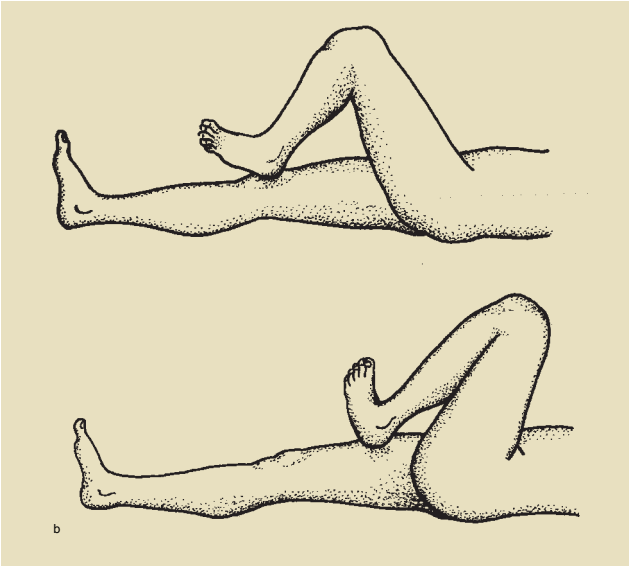
3. Index-nose test. The subject is instructed to, starting from the initial position of the previous maneuver, touch the tip of his nose, or the lobe of the opposite ear, with the end of the index finger of one arm, return to the starting position and perform the same with the other arm, that is, alternately, as quickly as possible, first looking and then with his eyes closed.

   The explorer will see if the subject can directly and quickly touch the indicated site with his or her finger; or if it also makes a series of glides and oscillations, touching other parts of the face before the one indicated.

4. Proof of the index, by Bárány. This test, actually called " index deviation" or "Bárány" test, is used with the objective of exploring the existence of deviation of the index towards the side of the altered labyrinth, but it can be used as another test of dynamic coordination (see the examination of the vestibular portion of the VIII nerve, in Chapter 15).

5. Heel-knee test. It is performed with the subject lying supine, never standing, and they are invited to touch the opposite knee with the heel of one foot. It is real-

Fig. 14.4 Heel-knee test: a, normal. The heel exactly touches the knee; b, pathological. The heel goes beyond the knee (hypermetria).



He runs alternately with both legs and as quickly as possible, first looking and then without looking. The explorer will carefully observe how the movement is carried out; whether the subject correctly touches the knee with his heel, or whether the foot first carries out a series of oscillations and glides before managing to touch the knee, and if once it has reached it, it cannot remain there except for a short time, and also how vision influences it, that is, whether the test is performed equally well when occluding the eyes (fig. 14.4).

The creativity of some semioticians has given rise to different variants of the classic tests, which are also valid. In this test, once the heel has reached the knee, it slides along the entire length of the anterior surface of the tibia, up to the ankle.

6. Diadochokinesia. With the subject seated and the palm of both hands resting on the thighs, he is ordered to rotate his hands simultaneously to touch the thighs with his palmar aspect alternating with his dorsal aspect, as quickly as possible, first with his eyes open and then, closing his eyes.

   This can be done without difficulty, if there is no alteration of dynamic coordination. If the subject has adiadochokinesia, he loses simultaneous coordination, especially when he closes his eyes, and it is observed that when one hand touches his palmar aspect, the other does so on his dorsal aspect, or they do not touch at the same time.

There are many other tests to explore kinetic or dynamic coordination, such as those explored with walking (see this section, at the end of Chapter 13). If you want to detect milder alterations in coordination, you can observe the performance of more complex and delicate acts, such as unbuttoning your shirt or threading a needle.

PRAXIA

Lapraxia was briefly defined in the previous chapter, when the exploration of language was discussed.

We will insist that it is the ability to more or less automatically carry out certain habitual systematic movements, for a specific purpose, which are known as intentional psychomotor acts or gestures.

These acts can be transitive or intransitive. Transitives are those that are executed through, or with the intervention of, objects (combing one's hair, buttoning or unbuttoning one's shirt, tying a shoelace, lighting a cigarette, brushing one's teeth, etc.). Intransitives are those that do not require the intervention of any object for their execution, such as the military salute,

make the sign of the cross, greeting the audience from a stage.

EXPLORATION OF PRAXIA

1. Transitive acts. Ask the person to perform one or more of the actions we mentioned above, or others in the same category, such as drinking a glass of water.

2. Intransitive acts. Tell the person to perform intransitive acts like those mentioned above, or others in the same category, such as showing anger or sticking out the tongue.

3. Imitative acts. Ask the subject to imitate the transitive or intransitive acts that you perform.

As can be seen, during the physical examination, before reaching the nervous examination, the person being examined has already performed innumerable transitive, intransitive and imitative acts; As examples we have: unbuttoning clothes, performing maneuvers that involve motor acts, ordered or by imitation, such as during the exploration of the SOMA or the taxi. You just have to keep it in mind. If they are performed normally, you can skip the scan when you get to this part of the exam and record each act as normal. If you detect any alteration, take it into account for a detailed examination at the time.

Praxia alterations are described in Volume 2, where this complex function is addressed in detail.

MOTILITY

GENERAL CONSIDERATIONS

Movement is a muscular activity governed by the central nervous system. Motility can be of two types (Table 14.1):

1. Kinetic motility or motility itself: it is what determines, through one or more muscular contractions, the movement of a segment, a limb or the entire body.

2. Static motility: it is that which, once the movement with displacement has finished, maintains the segment or the entire body, in the attitude to which the movement has taken it. This type of movement fixes, rather than displaces, and is nothing more than muscle tone

Kinetic motility is divided, in turn, into two large categories:

a) Voluntary active motility: this type of movement is executed through a volitional act, it is always preceded by the conscious mental representation of the movement that we want to execute, and the incitement is elaborated in cells located in the cerebral cortex and transmitted by its cylindroaxial extension, reaching the motor neuron of the anterior horn of the spinal cord or the motor nuclei of the cranial nerves located along the brain stem. The person in charge of carrying them out is the pyramidal or voluntary system.

b) Involuntary motility: it is that which is carried out independently of the will of the subject. In it there are several hierarchies of movements: reflex movements and automated movements. Reflexes, as we will see later, occur as a result of peripheral excitation, followed by an immediate motor response, which under normal conditions is always the same when faced with the same stimulus.

Example: striking the quadriceps tendon causes hyperextension of the leg. Automated movements are involuntary movements that accompany volunteers. Example: the swinging of the upper limbs when walking.

These involve a more complex nervous mechanism and are carried out by the extrapyramidal system.

Table 14.1

Motility type	exciter device	Executing device
a) Kinetic motility:		
- Reflex movement	reflex arc	NEURON
- Automatic movement	Extrapyramidal system	PERIPHERAL
- Voluntary movement	Pyramidal system	MOTOR
b) Static motility:		
- Muscle tone	extrapyramidal system Or cerebellum	

PRODUCTION MECHANISM

Anatomofunctional count

Voluntary active activity is carried out through the pyramidal pathway (Fig. 14.5), which constitutes the main efferent sector of the cerebral neocortex. It has its origin (60%) in the pyramidal cells or giant cells of Bets, located in each hemisphere, in the V layer of the motor cortex, which includes: the precentral gyrus or ascending frontal gyrus and the posterior 1/3 of the three frontal gyri (premotor cortex) and 20% of its fibers originate from the somatic sensory areas.

Experimental physiology has made it possible to demonstrate that in the motor area, the different centers that have, under their dependence, the movements of the different segments of the body, are represented, just as in the somatosensory area.

These centers are in a well-established order, which schematically is as follows:

a) The lower third corresponds to facial movements; Below it is the center for the movements of the tongue and lips.

b) The middle third, for the movements of the opposite upper limb.

c) The upper third and the paracentral lobe of the medial aspect of the cerebral hemispheres give rise to motor excitations destined for the opposite hemitroncus and lower limb.

At the foot of the inferior frontal gyrus of the dominant hemisphere (generally the left) lies the motor center of speech or Broca's center.

From this first motor neuron of the pathway originate the cylindroaxial extensions, constituting the pyramidal pathway, which are arranged in a fan shape in the cortical white matter, forming the corona radiata (oval center) and descend through the internal capsule, located between the gray nuclei at the base of the telencephalon. The pyramidal tract is made up of two bundles of fibers: the corticonuclear or geniculate bundle and the corticospinal bundle.

The fibers of the corticonuclear bundle or geniculate bundle (Greek: genus, knee) descend through the knee of the internal capsule and extend to the motor nuclei of the cranial nerves located along the brain stem.

Behind this, descending through the anterior two-thirds of the posterior arm of the capsule, runs the corticospinal bundle, with the fibers destined for the muscles of the upper limb located in its most anterior portion; and more caudally, those of the trunk and lower limb.

After passing through the internal capsule, the pyramidal bundle is located at the foot of the mesencephalic peduncle. From this point on, the corticonuclear bundle sends fibers to the motor nuclei of the contralateral cranial nerves, as they appear in the brainstem, and some fibers to the motor nuclei on the same side.

Consequently, the motor input leaves one hemisphere of the brain to go to the muscles on the opposite side.

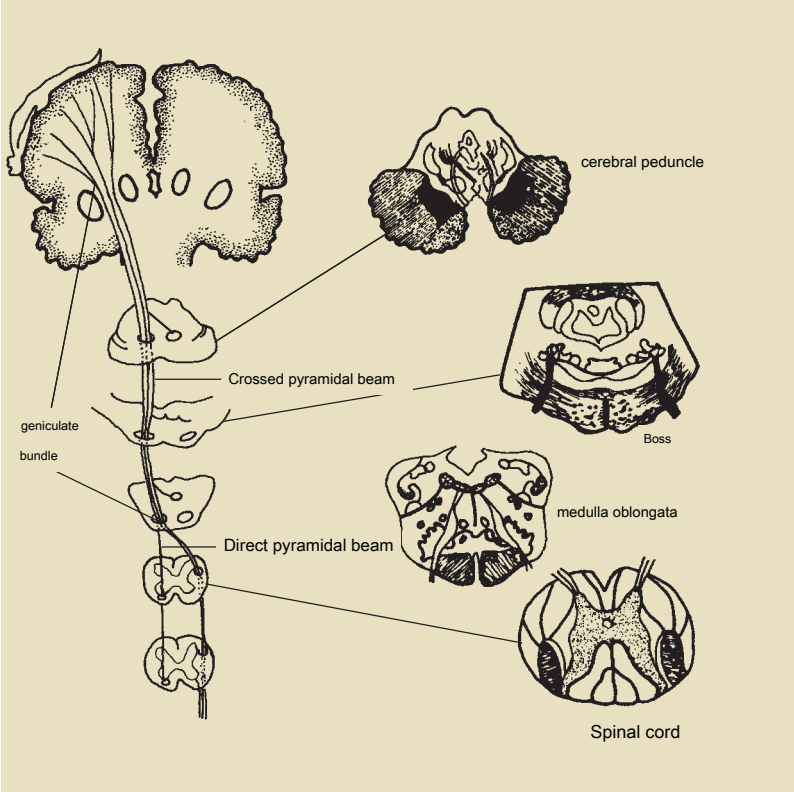


Fig. 14.5 Pyramidal pathway.

to. The fibers destined for the motor nucleus of the VII nerve or facial nerve have a special distribution, explained later with this nerve.

At the level of the pons or pons, the fibers of the pyramidal bundle are arranged in the anterior portion of this, in a dispersed manner, due to the presence of the fibers of the corticopontocerebellar bundle, meeting again at the level of the medulla oblongata, to constitute the pyramids.

At the lower limit of the pyramids, most of the fibers of the corticospinal tract cross the midline (pyramidal decussation) and descend along the lateral cord of the spinal cord forming the lateral or crossed corticospinal tract, which synapses on the alpha motor neuron of the anterior horn of the cord, also called the second neuron of the pathway or final common pathway of Sherrington. The rest of the fibers of the corticospinal bundle that were not decussated descend through the anterior cord on the same side as their cortical origin, forming the anterior corticospinal bundle; But as they descend through the spinal cord, they synapse on the contralateral motor neuron, so ultimately the entire bundle will be crossed.

The axons of the second neuron of the pathway, located in the anterior horn of the spinal cord or in the motor nuclei of the cranial nerves of the brainstem, emerge from the spinal cord or the trunk, respectively, constituting going the peripheral motor nerves that terminate in the muscle (see fig 14.5).

EXPLORATION TECHNIQUES

Voluntary active motility

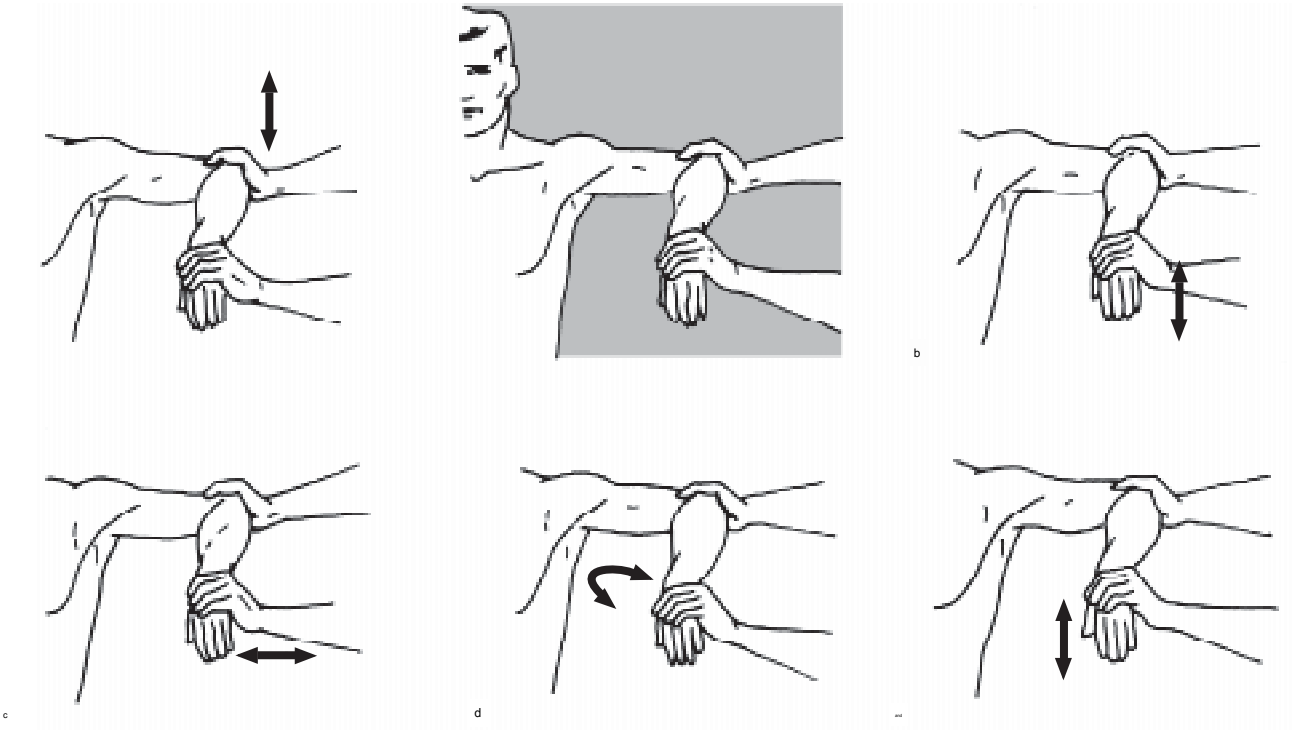
Voluntary active motility is explored by asking the subject to perform with each and every part of their body all the movements that correspond to each of the joints. Naturally, in order to assess whether there is any limitation in them, it is essential to know in advance what the normal movements of each of the joints in our body are.

It will be observed, while the individual performs said movements, the way in which they are executed, the greater or lesser difficulty with which they are performed and whether similar movements corresponding to similar portions, on opposite sides of the body, are carried out in the same way.

The exploration of voluntary active motility is completed with the determination of segmental muscle strength, for which there are various maneuvers.

A very simple one consists of the doctor giving both hands to the subject, and after ensuring by the way in which the subject gives him the hands, that he cannot do anything.

Fig. 14.6 The center figure shows the initial position of the subject; This is maintained and offers resistance to the force exerted by the examiner. The following are explored: a, separation, elevation and approximation of the arm; b, arm rotation; c, elbow flexion and extension; d, supination and extension of the forearm; e, flexion and extension of the wrist.



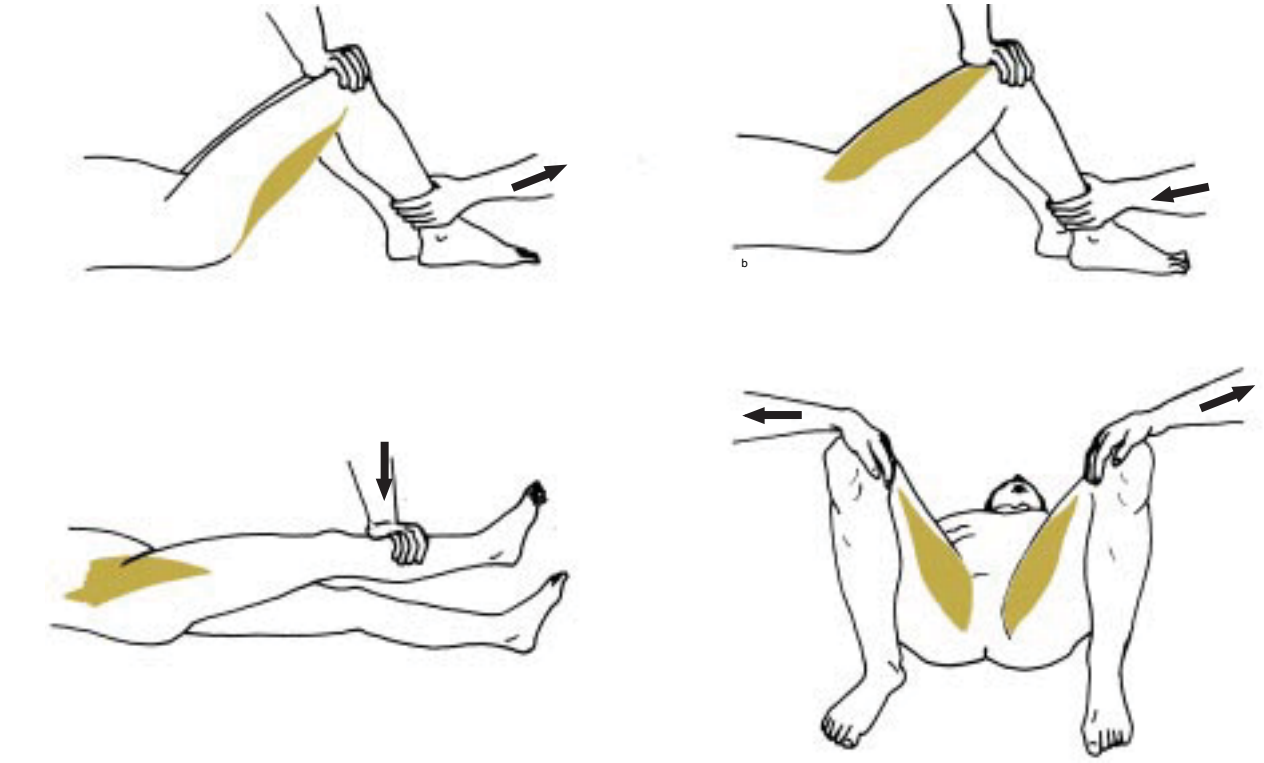
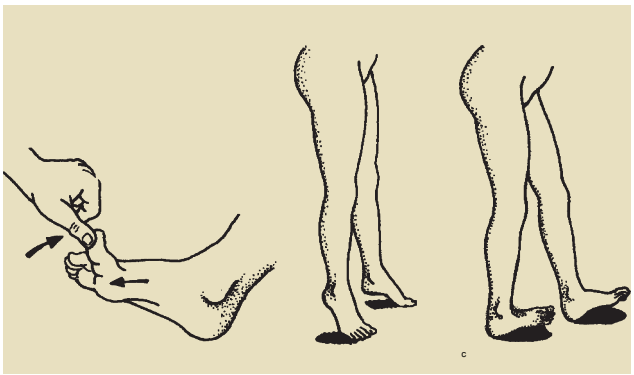


Fig. 14.7 Muscle examination: a and b, of the pelvic girdle muscles;
C, iliac psoa: the subject raises the extended leg against the pressure exerted in the opposite direction by the examiner; d, of the adductors of the thigh.

damage, asks him to squeeze his hands tightly, with which he is already determining the segmental muscle strength of the two upper limbs simultaneously.

The segmental muscular strength of any part of our body can be explored by asking the subject to perform a movement with said part, and by opposing that movement (figs. 14.6, 14.7 and 14.8), he confirms in this way the force with which the subject performs it, also checking whether similar movements carried out with portions of one side of the body are made with the same intensity on the opposite side (see also the physical examination of the body). SOMA).

Fig. 14.8 Assessment: a, of the strength of dorsiflexion and plantar flexion of the foot and big toe; b, ambulation on the balls of the feet; c, ambulation on heels.



There are maneuvers that allow segmental muscle strength to be studied with an objective and not a subjective criterion, such as the one previously described. They are those of Barré and Mingazzini.

Barré maneuver. (For the lower limbs.) Subject in a prone position, legs at right angles to the thigh (that is, perpendicular to the surface of the bed). You are instructed to keep them in that position as long as you can, recommending that they do not touch each other (fig. 14.9).

If one of the limbs is affected in its motility, it will fall before the other, sometimes slowly or abruptly until it reaches the plane of the bed. It is possible to observe some "shakes" through which the subject tries to recover the lost initial position.

Mingazzini maneuver of lower limbs. It is explored with the subject in a supine position. He is ordered to flex his thighs at an angle of 90° in relation to the trunk and both legs without touching the knees, also at an angle of 90°, in relation to the thighs. The subject is encouraged to keep them in that position for as long as possible and it is observed whether one limb falls first than the other, which is pathological, or whether both limbs remain at the same level or fall slowly at the same time, as the subject loses strength, as normally occurs.

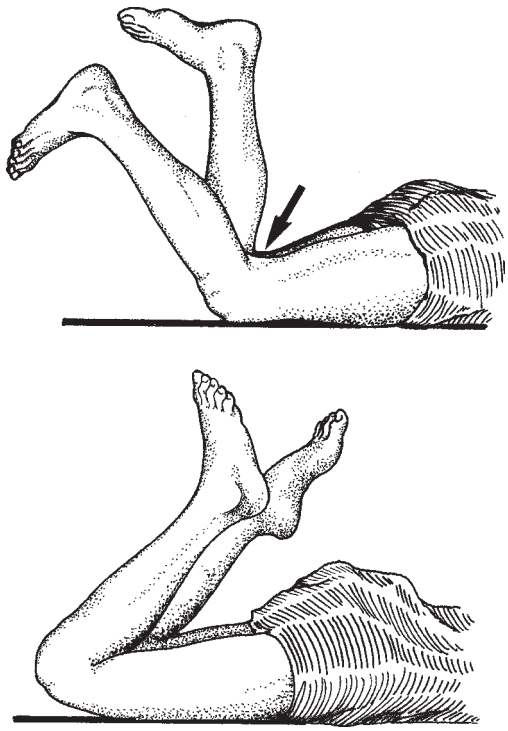


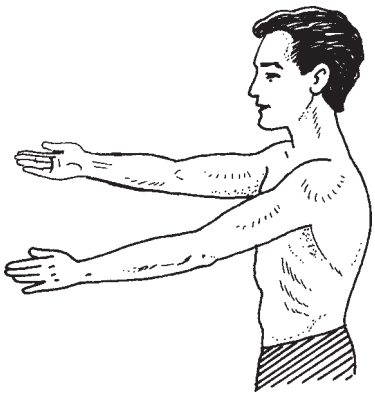
Fig. 14.9 Barré maneuver for the lower limbs.

Mingazzini maneuver of upper limbs. The person is invited to keep both upper limbs extended with the back of their hands facing up (although down is preferred). Zimman advises that to perform this test the subject keeps his eyes closed. When there is a pyramidal deficit, it is observed that, depending on the intensity, first there is pronation of the hand, then it descends, then the forearm and, finally, the entire limb (fig. 14. 10).

Involuntary active motility

It should also be observed, whether they exist or not, involuntary active movements, such as tics, tremors, seizures, etc., which are pathological.

Fig. 14.10 Mingazzini maneuver for the upper limbs.



MUSCLE TONE

CONCEPT

By muscle tone we understand a permanent, involuntary contraction, of variable degree, non-fatiguing, of a reflex nature, aimed either at maintaining an attitude or at keeping the muscle ready for a subsequent voluntary contraction; It can be defined, therefore, as the involuntary permanent tension of the core that is voluntarily relaxed.

PRODUCTION MECHANISM

Normally every muscle, at any time, has a certain amount of activity or tension that serves mainly to maintain posture. This constant state of tension of the muscle fascicles is what constitutes muscle tone, which is created in the muscle itself through the so-called myotatic reflex (from the Greek myós:

muscle; stasis, tension), also called the Liddel-Sherrington extension reflex, that is, the contraction of a muscle due to its passive elongation. This reflex originates in the neuromuscular spindles located in the thickness of the muscle, which are very sensitive to stretching, and when stimulated by the lengthening of the muscle fibers located around them, they transmit this stimulation to the motor cells of the anterior horn of the spinal cord (fibers and gamma neurons of the same). From these neurons motor stimuli come out that go to the endings of said nerves in the muscle, to maintain the particular or special state of tension that constitutes muscle tone.

The medullary center of the tone is part of a simple reflex arc. Higher centers or structures act on this spinal center, some of which are dynamogens or facilitators of tone, while others are inhibitors or brakers of it. These higher centers reach, through different routes, the motor neuron of the anterior horn of the spinal cord, the true "crossroads" of the entire motor system, and from where the motor nerve, also called trillum, or final path common to all the motility pathways, both dependent on the corticospinal and pyramidal pathways (see "Motility"), and the extrapyramidal pathways, originates.

The existence of two types of motoneurons has been demonstrated: the alpha neurons, which are located in the anterior horn of the spinal cord, are phasic and tonic, and have different anatomofunctional characteristics, but are related to active muscle fibers, that is, those that produce movement. The gamma motoneurons, which, on the contrary, innervate the afferent traction receptors (origin of the myotatic reflex), are sensitive to pain, but do not intervene in active contraction. They are responsible for maintaining muscle tone.

The higher centers that act on the anterior horn neuron are: cerebral cortex, gray nuclei of the base, cerebellum, reticular substance and vestibular centers and pathways, which inhibit or facilitate tone.

To understand how these centers act on muscle tone, it is necessary to keep in mind that damage to a tone-inhibiting center will be manifested by an increase in tone; while the lesion of a facilitating or dynamogenic center will manifest itself clinically by a decrease in it.

EXPLORATION TECHNIQUES

1. Inspection. First we will observe the appearance and attitude of the extremities; whether the muscle masses maintain their normal appearance and relief or whether, on the contrary, the relief is increased or decreased, for which, in cases of unilateral injuries, it is useful to compare the muscles on one side of the body with those on the other.
   The inspection of the trunk muscles and observing their relief should not be omitted.
2. Palpation. All the muscles of the body should be examined during this maneuver, especially those that during the inspection we suspect have altered tone. When performing palpation, it should be taken into account that the "normal" degree of consistency or hardness of a muscle will depend, among other things, on the muscular development of the subject. It should not be expected that a muscle from a person who does not perform any muscular activity will have a consistency similar to that of someone who is constantly training their muscles. Thus, what in one case may seem like an increase in tone may not be, and vice versa.

3. Resistance of the muscles to manipulation or passive movement. This is the procedure of choice for exploring muscle tone. To do this we ask the subject to try to maintain as much muscle relaxation as possible, we take his extremities one by one and perform each and every one of the possible joint movements with it. We especially explore the passive flexion and extension of the limbs, the trunk, and the head. It is very important that during the execution of these movements the patient does not intervene voluntarily, because if this occurs, the motility being explored would cease to be passive and become active and, therefore, we would not be exploring muscle tone.

Special pitch maneuvers

Andre Thomas Passivity Tests

There are multiple, we will only describe one of them. It is based on the fact that the member that has its muscles hypotonic (decreased tone) when passive movements are imposed, it presents greater oscillations than the healthy one. Thus, if a subject who has hypotonia of the muscles of one arm is placed in a standing position, is held by the trunk and a rotation movement is given, so that his arms oscillate, it will be seen that the arm on the diseased side swings more widely than the arm on the healthy side.

Maneuvers in search of hypertonia due to meningeal irritation

There are several maneuvers in search of hypertonia due to different causes. But due to their importance, and because they are also part of the usual physical examination of the nervous system, we will describe the main maneuvers for exploring the meningeal sphere and which are positive when there is reflex hypertonia or rigidity of certain muscle groups, due to the irritation caused by inflamed meninges (meningitis).

Kernig maneuvers

Kernig maneuvers can be negative or positive. When a Kernig maneuver is positive it is because the Kernig sign was detected.

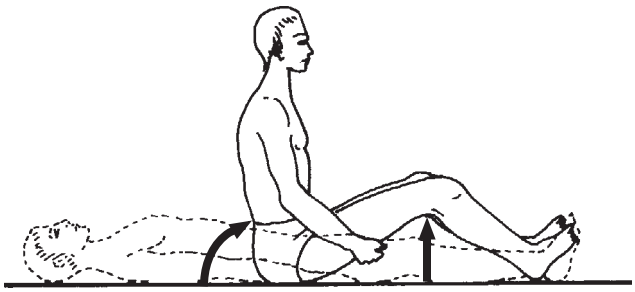
Kernig's sign can be looked for in two ways:

1. With the subject lying supine, he is made to flex his body until he sits up, passively lifting it, with the forearm and the examiner's hand behind and below the shoulders, while paying attention to what is happening in the lower limbs.

   If the maneuver is negative, the lower limbs do not flex or there is a slight flexion of the knees at the beginning. If the maneuver is positive, that is, there is Kernig's sign, when performing it the thighs are flexed over the pelvis and the legs over the thigh (fig. 14.11);
   Then, the free hand is rested on the knees, to try to obtain the extension of the lower limbs, which is impossible.

2. Lower Kernig maneuver. Kernig's sign is also investigated with the subject in a supine position and a lower limb is slowly raised (in supine flexion).

Fig. 14.11 Kernig's sign: when trying to passively raise the subject lying down, his knees are flexed.



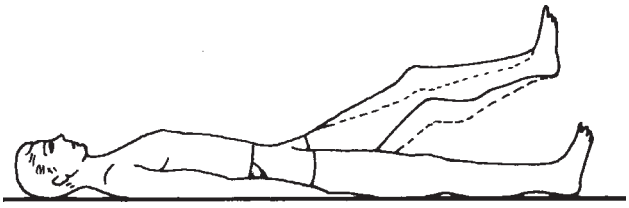


Fig. 14.12 Kernig's sign: when trying to passively raise the subject lying down, his knees are flexed.

hip) in a manner analogous to the Lassègue maneuver. When the limb has been raised to a certain height, there is a Kernig sign, if a flexion occurs in the knee joint, which becomes invincible and sometimes painful (fig. 14.12).

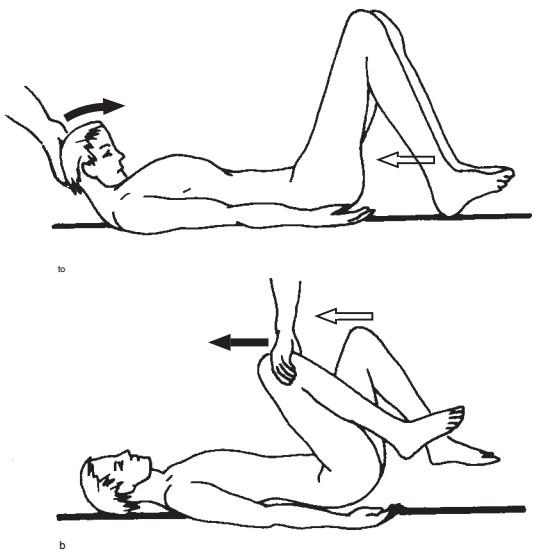
Brudzinski maneuvers

Two Brudzinski signs have been described: the neck sign and the contralateral reflex.

1. The upper Brudzinski or neck sign is investigated in the following way: one hand is fixed on the subject's chest, to prevent it from rising, while the other tries to flex the head, and the examiner focuses his attention on the lower limbs. If the sign exists, the lower limbs are flexed at the knee and hip (fig. 14.13 a).

2. The contralateral Brudzinski reflex consists of the following: if one thigh is strongly flexed over the pelvis, it is observed that the opposite limb reproduces the movement (fig. 14.13 b).

Fig. 14.13 Sign of Brudzinski maneuvers: a, the subject's neck is vigorously flexed and both lower limbs are observed to be flexed; b, when the right lower limb is flexed, the homologous limb spontaneously flexes, which constitutes the contralateral Brudzinski reflex.



to

b

TROPHISM

The exploration of trophism has been addressed throughout the physical examination, fundamentally of the skin and the osteomyoarticular system. Trophism alterations are explained in Volume 2.

REFLECTIVITY

CONCEPT

The reflex is nothing more than the motor or secretory response, independent of the will, provoked immediately after the application of a sensitive or sensory stimulus, which may or may not be conscious.

If food reaches the stomach, it immediately causes the secretion of gastric juice. In this case the response has been to an unconscious stimulus, because the person does not know when the food reached the stomach.

If we touch a burning surface with any part of our body, we immediately remove the part of the body from contact with the surface. In this case the stimulus is conscious.

The reflexes that we have exemplified and those that we will study in this chapter are all unconditioned or natural reflexes (with the exception of pathological responses) and many of them are carried out without intervention of the cerebral cortex. The conditioned reflexes discovered by Pavlov always have cortical participation and although they are of great importance for learning, they do not enter into neurological exploration.

PRODUCTION MECHANISM

Anatomically, the reflex arc, in its simplest form, is made up of: afferent sensory fibers, which collect the stimulus initiating the reflex and take it to a center from which motor or secretory fibers originate, which conduct the stimulus to a muscle to contract or to a gland to secrete. Generally, the center of the reflex arc consists of a third neuron, called the intercalary or internuncial neuron, which links the sensory cells where the sensory fibers end, with the motor cells that give rise to the motor or secretory fibers (Fig. 14.14).

The integrity of the reflex arc is essential for the reflex to occur, from which it follows that the absence of a reflex in the face of the appropriate stimulus to produce it may be due to:

1. Interruption of the sensory pathway responsible for perceiving the stimulus.
2. Alteration or damage to the reflex center, including alteration of the neuron or intercalary neurons it has.

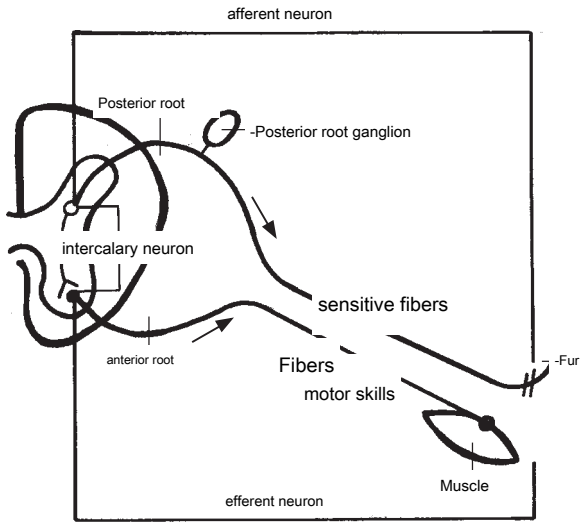


Fig 14.14 Reflex arc.

3. Interruption in the motor or secretory pathway responsible for carrying the stimulus from the center to the effector organ, muscle or gland.
4. Impossibility of the effector organ to carry out the movement or secretion that was ordered. A person who has an ankylosis of one knee will not be able to extend the leg on the thigh, which is caused by percussing the tendon under the kneecap. This response would constitute an areflexia if it were not due to functional impotence of the knee.
   Likewise, if the gland in charge of making a secretion is destroyed, it will not be able to produce it even if the entire nerve pathway is intact.

The reflex centers are located, in general terms, at the entire height of the neuraxis, therefore, the examination of the reflexes serves to determine the integrity or not of the centers and, consequently, locate the site and height of a neuraxis lesion.

Reflexes are classified into:

1. Osteotendinous or deep reflexes.
2. Cutaneomucosal or superficial reflexes.
3. Spinal automatism reflexes.
4. Posture and attitude reflexes.

EXPLORATION TECHNIQUES

Osteotendinous or deep reflexes

Osteotendinous or deep reflexes are understood to be those in which the response is obtained by applying a mechanical stimulus (blow with the reflex hammer) to the tendons and occasionally to the bone or periosteum. By custom, they are called tendon reflexes, periosteal reflexes, etc., because the stimulus is mediated through the elements. deep sensory reflexes (tendon or muscle spindles), therefore they should be considered proprioceptive reflexes. They are actually "muscle stretch reflexes" according to the physiological conception of Liddel and Sherrington.

To achieve a good response to the stimulation that aims to provoke a reflex of this type, it is essential ble:

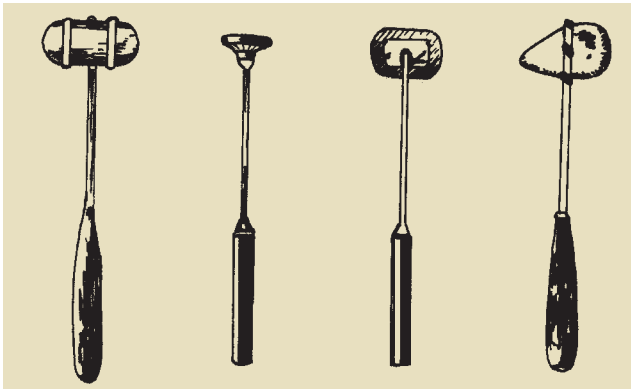
1. That the stimulus is intense enough, without being exaggerated.
2. That it is quick and that it is applied exactly in the right place for the reflection that it is trying to produce.
3. That the subject is in a state of adequate relaxation of the muscles corresponding to the reflex being explored.

We will try to achieve complete relaxation first and then, through passive movements, an appropriate degree of tension, which is obtained by placing the muscle that will respond, in an intermediate position between its maximum and minimum elongation. Let us clarify that this is a position that the explorer must achieve from the subject, who must remain totally relaxed during the exploration.
Sometimes this is not easy. Later we will describe the Jendrassik maneuver, intended to achieve muscle relaxation. It is also useful to talk to the person while being examined, in order to distract their attention from the maneuvers intended to find the reflexes.

The reflections will be explored symmetrically, first on one side and then on the other to compare the results. Normally, the responses are symmetrical.

A special hammer of which there are several models is used in the examination (fig. 14.15). This hammer is characterized because the striking part is made of rubber and the handle is usually made of metal or wood. It should be taken by its base, with the most skillful hand, and struck gently.

Fig. 14.15 Various types of hammers used in the examination of reflexes.



Technique for using the percussion hammer

The most commonly used hammer is the one with the rubber part through which it is struck, triangular in shape, with a pointed end for small tendons and a blunt end for large tendons.

Hold the hammer between your thumb and index fingers, by the final third of the handle, and allow the hammer to swing quickly and freely downward, moving your wrist as in finger drumming. Gripping the hammer with the whole hand, or very close to the striker rubber, inhibits free movement.

Next we will study the most important bone tension reflexes:

- From the cephalic portion.

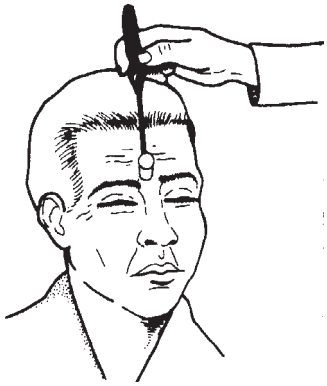
Of the upper limbs.

Of the lower limbs.

Osteotendinous reflexes of the cephalic portion

1. Orbicularis oculi reflexes. Superciliary and nasopalpebral (fig. 14.16). Percussing the superciliary arch and the root of the nose while the subject is with his eyelids half-closed, causes contraction of the orbicularis oculi and therefore bilateral palpebral occlusion (even if it is percussed on only one side).
   It is advisable to perform them with your eyes closed, so that the person does not see the percussive hammer, preventing the contraction from occurring as a threat reflex and not due to percussion.
   Afferent pathway: V pair (supraorbital branch).
   Efferent route: VII par.
   Reflexogenic center: bridge.
2. Masseteric reflex. It can also be called mandibular (the masseter and temporalis muscles are involved). The subject remains with his mouth half open and in that position the chin is tapped directly with the hammer or the index finger of the left hand is placed transversely under the lower lip, either

Fig. 14.16 Orbicularis oculi reflexes: nasopalpebral reflex.



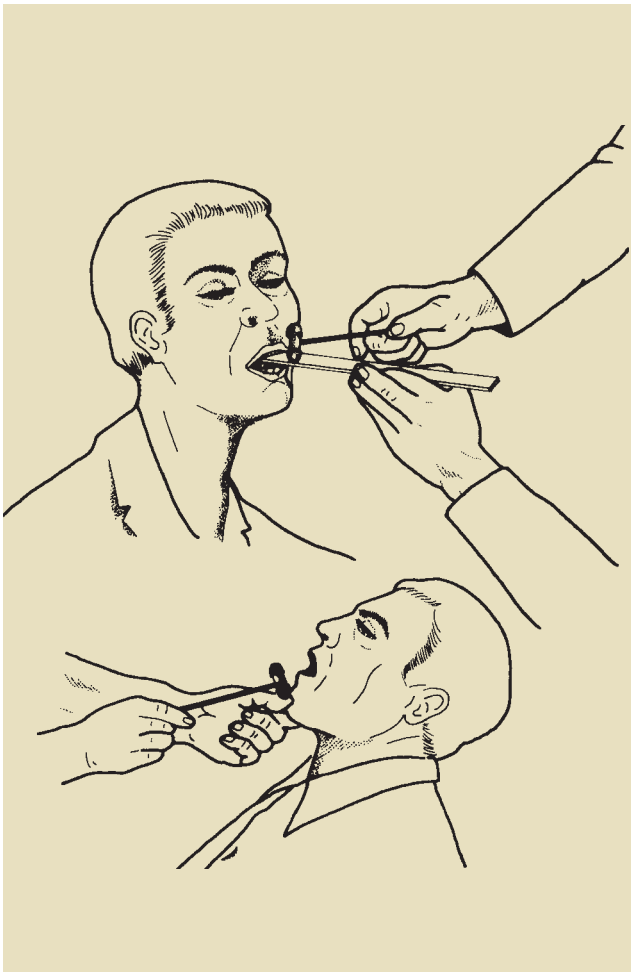


Fig. 14.17 Masseteric reflex: two ways to explore it.

rested against the jaw, and is percussed on it.

A tongue depressor can also be inserted into the mouth, resting on the lower dental arch and percussing on it. The answer is elevation of the jaw (Fig. 14.17).

Afferent pathway: V pair (supraorbital branch).

Efferent pathway: V pair (motor branch).

Reflexogenic center: bridge.

Osteotendinous reflexes of the upper limbs 1. Bicipital reflex.

Keep the subject's forearm semi-flexed and semi-supinated, resting on your own supported by the elbow, or resting on the thighs, if the subject is sitting, or on the trunk, if lying down. The explorer rests the thumb of his free hand on the subject's biceps tendon, in the antecubital fossa and strikes on the thumbnail, or on it, with the thinnest part of the percussive hammer, if it is triangular in shape. Flexion of the forearm on the arm is obtained (fig. 14.18).

Reflexogenic centers: cervical spine (CS and C6 segments).

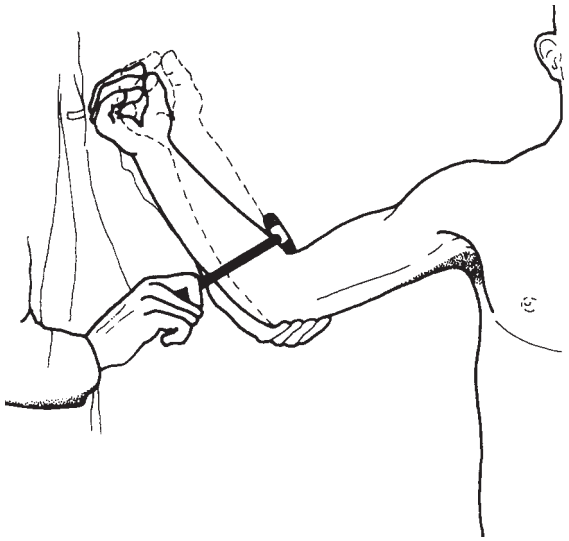


Fig. 14.18 Bicipital reflex.

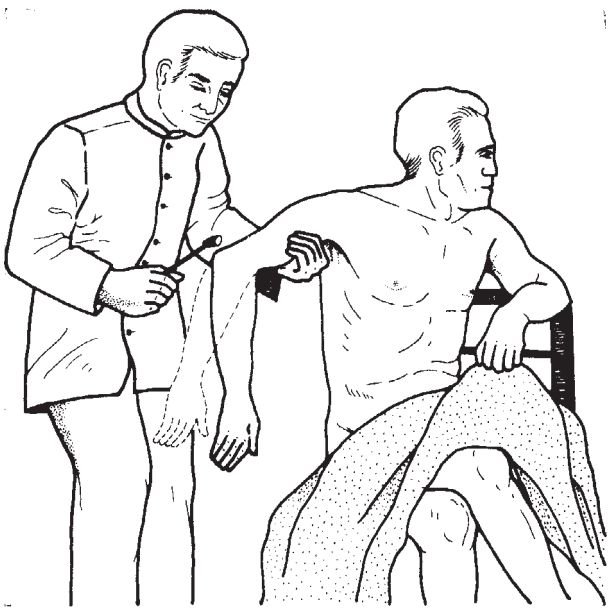
2. Tricipital and olecranon reflex. With one hand, the subject's forearm is taken by the elbow and held on his forearm, crossing the thorax, placed at right angles to the arm and the triceps tendon is percussed (taking care not to percuss the olecranon), preferably with the wider side of the hammer. The response is the extension of the forearm over the arm (tricipital reflex). Another alternative is for the forearm to hang freely at the side of the body, supporting the arm, in 90° abduction (fig. 14.19).

Reflexogenic centers: segments C6, C7 and C8.

If the olecranon is percussed, the response is flexion of the forearm over the arm (olecranon reflex).

Reflexogenic centers: cervical segments C5 and C6.

Fig. 14.19 Tricipital reflex.



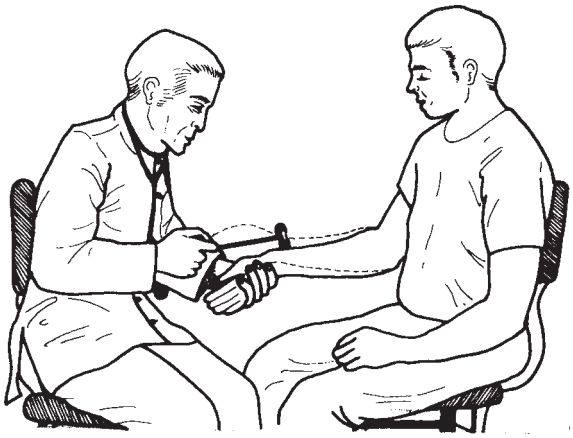


Fig. 14.20 Long supinator reflex.

3. Long supinator or brachioradialis reflex. Misnamed " styloradial". since it is a reflex of "muscle stretching" and not osteoperiosteal.

The upper limb is placed with the forearm semi-flexed on the arm, so that it rests by the ulnar edge of the forearm on the palm of the explorer's hand, or on the subject's legs.

The styloid process of the radius, through which the supinator longus tendon passes, is then percussed. The main response is flexion of the forearm; The accessory response is a slight supination and flexion of the fingers (Fig. 14.20).

Reflexogenic centers: C5 and C6.

4. Ulnopronator reflex. With the upper limb in the same position as that indicated for the long supinator reflex, the doctor lightly percusses the styloid process of the ulna, tangentially from top to bottom; The answer is pronation. This reflex is almost always weak and only its unilateral abolition or when it becomes very evident, in cases of hyperreflexia, has value (fig. 14.21).

Reflexogenic centers: C7 and C8.

5. Finger flexor reflex. The forearm in semiflexion and supination with the last phalanges of the fingers in slight flexion (thumb in extension). This can be done in two ways: the examiner percusses the subject's flexor tendons in the carpal canal or above; or, on the contrary, he places his middle and index fingers on the palmar surface of the last phalanges of the last three or four fingers of the subject and performs percussion on them. The answer is the flexion of the last four fingers. Sometimes thumb flexion is included.

Reflexogenic center: C8.

There are two techniques to explore other variants of this reflex: Troemner's and Hoffmann's.

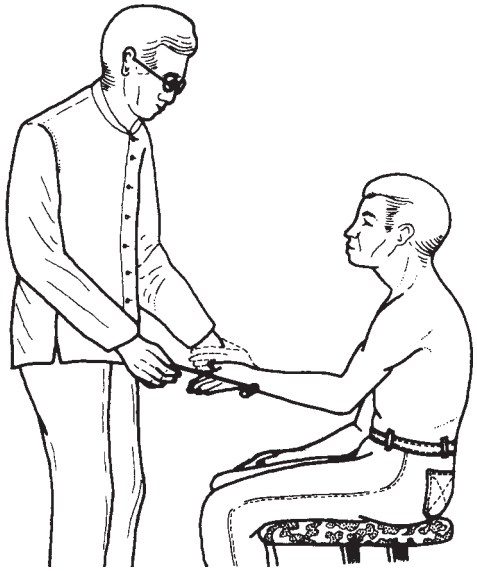


Fig. 14.21 Ulnopronator reflex.

In the Troemner technique the subject's forearm is placed in semiflexion and pronation. The doctor percusses the pad of the middle and index fingers with his fingers.

The answer is finger flexion.

In the Hofinann technique, the subject's hand is placed pronated. The examiner takes the terminal phalanx of the middle finger between his index finger and thumb, and, using a sudden movement, flexes the last phalanx (compressing the nail) and quickly releases it. The positive response consists of a sudden jerk of the thumb in adduction and flexion, with or without flexion of the other fingers.

This reflex is identical to that of the finger flexor, which we saw previously. For some it would be an index of muscle hypertonia rather than pyramidal injury. It is not always positive in such lesions, and, on the other hand, it can be found in nervous people without neurological organic lesions.

The response and center are the same as the previous reflex (C8).

Barraquer Bordas insists that these three reflexes are normal, variable in the magnitude of their responses from one individual to another and that, therefore, it cannot be stated, as has often been done, that the Hoffmann reflex or the Troemner reflex have the same significance as the Babinski sign, which would imply an unsustainable criterion and could lead, in practice, to serious errors.

Osteotendinous reflexes of the lower limbs 1. Middle pubic reflex (Guillain and Alojouanine). The person should be placed supine with the thighs apart and the legs slightly bent. It is then percussed on the pubic symphysis. The answer is twofold: a higher one, which consists of the contraction of the abdominal muscles, and another lower one, which is the approximation of both thighs, by the contraction of the adductors of both limbs.

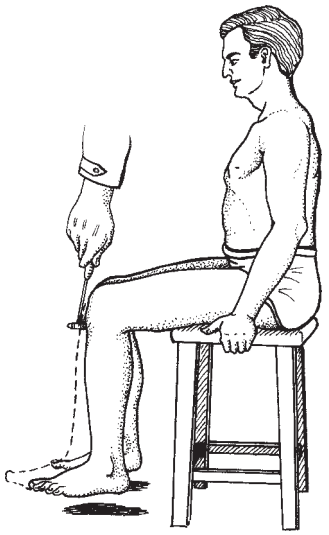


Fig. 14.22 Patellar reflex: examination in sitting position.

Reflexogenic centers: corresponds to the segments between D10 and LI (D10, D11, D12 and L1).

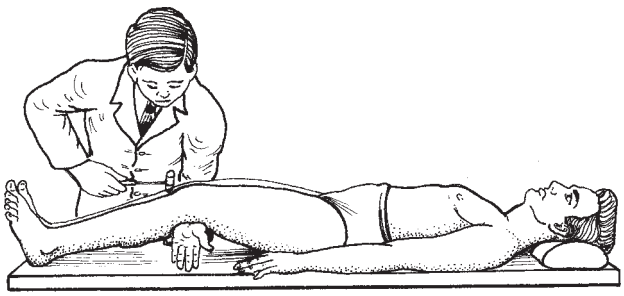
Its elevation with simultaneous abolition of the cutaneous-abdominal reflex (see below) is an obvious sign of injury above D6. The abolition of both reflexes indicates that the lesion is below this level.

2. Patellar or patellar reflex Quadriceps reflex. The technique can be:

a) Subject sitting in a chair or on the edge of the bed, with feet hanging (fig. 14.22). It is struck directly on the patellar tendon. The answer is leg extension.

b) Subject in bed (fig. 14.23). The lower limbs are raised slightly with one hand placed under the popliteal fossa, thus achieving a discreet flexion of the leg on the thigh, leaving the knee high. The patellar tendon or quadriceps tendon is percussed.

Fig. 14.23 Patellar reflex: examination in the lying position.



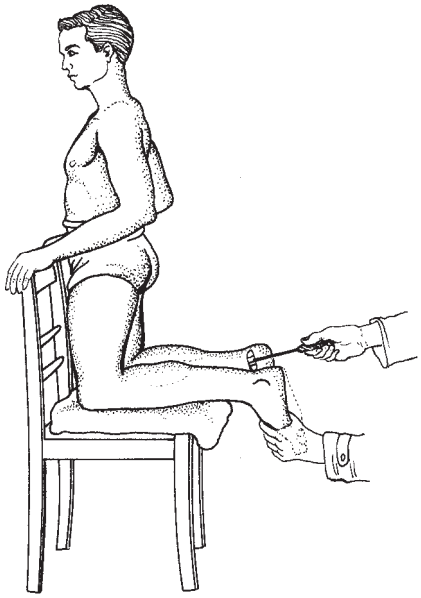


Fig. 14.24 Achilles reflex.

The answer is leg extension.

Reflexogenic centers: located in L2, L3, L4.

3. Achilles reflex. Triceps surae reflex (fig. 14.24).

The exploration can be carried out in three different ways:

a) Seated subject: limbs hanging over the edge of the bed, stretcher or chair; Lift the foot slightly with one hand and tap the Achilles tendon with the other, taking care not to tap the calcaneus.

b) Subject kneeling on the bed, stretcher or chair, feet outside the edge: the sole of the foot is brought slightly forward and the Achilles tendon or calcaneal tendon is struck.

c) Subject lying down: the foot of the lower limb to be explored is passively placed on the opposite one in semiflexion and abduction, resting on its external malleolus; With one hand, take the sole of the foot and hold it in slight flexion; the tendon is percussed.

The answer is foot extension.

Reflexogenic centers: L5, S1, S2.

Jendrassik maneuver. When the aforementioned reflexes, especially the patellar, are not obtained, the Jendrassik maneuver can be tried in order to achieve greater muscle relaxation. The subject is told to place the fingers of his left hand in a downward claw formation in the gap formed by the fingers of his right hand, which should form an upward claw, and then try to pull as if he wanted to see which hand has more strength. While the subject pulls hard on his hands, the tendon is percussed. Often, a reflection that was not achieved before is obtained in this way (fig. 14.25).

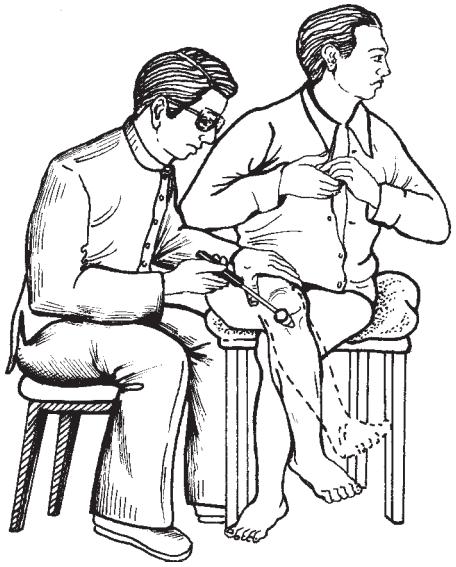


Fig. 14.25 Jendrassik maneuver.

Cutaneomucosal or Superficial reflexes

Superficial or exteroceptive reflexes are understood to be those obtained in response to the application of a stimulus, either on the skin or on the mucous membranes. For this purpose, a common needle or a pin is used (for cutaneous exploration) and cotton when exploring the mucous membranes.

These are reflex arcs of spinal, multineuronal, polysynaptic integration, whose starting point depends on a nociceptive stimulus. Hence they are also called nociceptive reflexes. We are going to expand somewhat from the general point of view and in the light of clinical and electromyographic interpretations, especially from the Swedish school, although for the semiological descriptive part we will faithfully follow the classic descriptions.

By studying in normal individuals the nociceptive reflexes of the lower extremities, clinically called defense reflexes by Babinski, the following facts have been verified:

1. That such reflexes are coordinated movements."
2. That the type of response externalizes a movement of escape from the stimulus.
3. That cutaneous-abdominal reflexes are an integral part of the spinal nociceptive defense mechanism.

These facts open a new field to adequately interpret many pathology issues, but, as we said at the beginning, we are going to describe these nociceptive reflexes according to the classics:

1. Corneal and conjunctival reflex. Stimulation of the cotinea and bulbar conjunctiva with a handkerchief (angled tip) or a small piece of cotton causes contraction of the orbicularis oculi. It is necessary

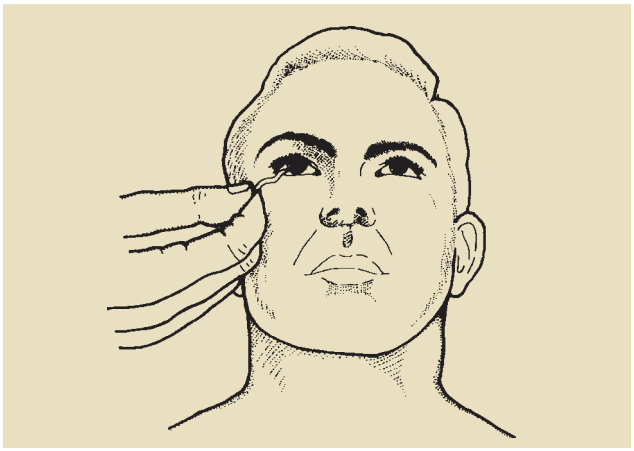


Fig. 14.26 Corneal or conjunctival reflex.

It is necessary to introduce the cotton laterally from outside the subject's visual field to suppress the defensive reflex (fig. 14.26).

Afferent route: V pair (ophthalmic branch).

Efferent pathway: VII pair.

Reflexogenic center: bridge.

2. Pharyngeal or gag reflex. By exciting the soft palate or the posterior wall of the pharynx (with a swab), contraction of the pharyngeal constrictors occurs, accompanied by nausea.

Afferent route: IX pair.

Efferent pathway: X pair.

Reflexogenic center: bridge.

3. Abdominal cutaneous reflexes. It cannot be explored in obese subjects or those with flaccid walls. The person should be supine and with their lower limbs slightly flexed.

In this position, the abdominal area is excited with a pin. There are three reflexogenic zones: upper cutaneous-abdominal (or spigastric), middle abdominal (or umbilical) and lower (or hypogastric):

a) The upper cutaneous-abdominal reflex is sought by gently and quickly exciting the abdominal wall from the inside out or from the outside in, following a line parallel to the costal margin.

b) The average cutaneous-abdominal reflex can be discovered by horizontally exciting the abdominal wall, starting from the navel (that is, from the inside out) or from the outside in (reaching the navel).

c) The lower cutaneous-abdominal reflex is investigated by exciting the abdominal wall, on a parallel line, above the inguinal line (it can be from the inside out or from the outside in).

The response in all three cases is a contraction of the abdominal muscles, retraction towards the same side of the white line, and retraction of the navel, also ipsilateral.

Reflexogenic centers: upper cutaneous-abdominal (D7, D8 and D9); middle cutaneousabdominal (D9, D10 and Dll) and lower cutaneousabdominal (D1l and D12).

4. Cremasterian reflex and homologous reflex in women. In men, by exciting the inner surface of the thigh, in its upper part, or by compressing the mass of the adductors, the cremaster contracts, producing elevation of the testicle; There is also a contraction of the greater oblique or external oblique. With this same technique, contraction of the greater oblique (Geigel's reflex) is observed in women.

Reflexogenic centers: Ll and L2.

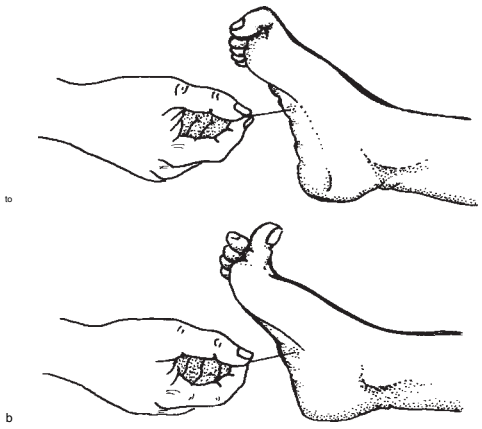
5. Plantarcutaneous reflex. Excitation with the pad of the finger, a pencil or a pin on the sole of the foot, on its external, middle or internal side, with a lot, light or very little pressure (depending on the circumstances), causes plantar flexion of the toes; It is the plantar reflex.

But in certain conditions, instead of flexing the toes, the big toe extends and the others flex, or they fan out. This phenomenon constitutes the Babinski sign (fig. 14.27), name of the great French neurologist who made it known in a communication of just 30 lines to the Society of Biology of Paris, on February 22, 1896. This reflection allows us to affirm the organic or functional alteration of the pyramidal pathway.

The Babinski response is normal in children in the first years of life (1 and 2 years) when the pyramidal tract has not yet been myelinated. But its presence is always pathological above this age, and is a sign of injury or dysfunction of the pyramidal pathway as we have pointed out.

The search for the Babinski sign requires certain thoroughness. Given its importance, it is necessary to proceed with method and patience before denying that it

Fig. 14.27 Cutaneoplantar reflex: a, normal; b, pathological (Babinski sign).



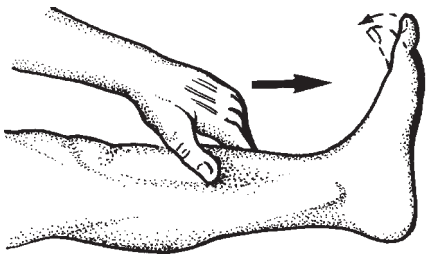


Fig. 14.28 Oppenheim maneuver to obtain the Babinski sign.

When the maneuver to investigate the Babinski sign results in abduction of the fifth finger, it is called the Poussep reflex.

6. Babinski substitutes. The same response (extension of the big toe) can be obtained through other maneuvers described below and which constitute substitute signs:

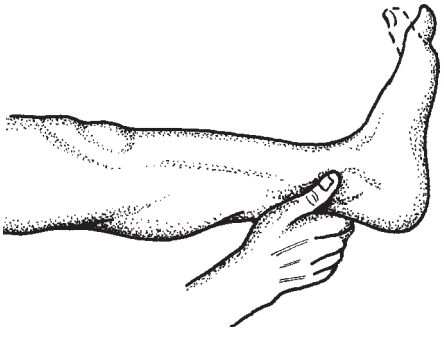
a) Oppenheim maneuver: this is called when the extension of the big toe is obtained by pressing the inner surface of the tibia from top to bottom with the thumb (fig. 14.28).

b) Schäffer maneuver: the same response is achieved by compressing the Achilles tendon (fig. 14.29).

c) Gordon maneuver: the same response is obtained by compressing the muscle masses of the calf (fig. 14.30) .

In reality, none of these maneuvers are as sensitive as the Babinski technique. Wartenberg says that after seventy years a better one remains to be discovered.

Fig. 14.29 Schäffer maneuver to obtain the Babinski sign.



Evaluation and recording of reflexes

It is of utmost importance that we point out that neither the total absence of reflexes nor their most exaggerated response are, in themselves, a sign of abnormality. Occasionally

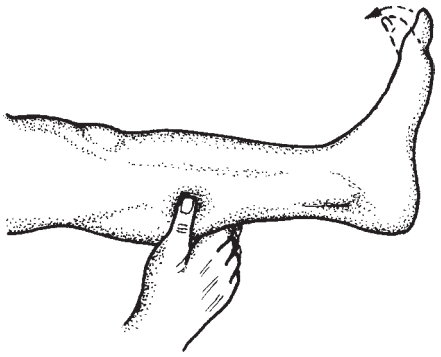


Fig. 14.30 Gordon maneuver to obtain the Babinski sign.

You can find people with areflexia who are otherwise normal. There have also been cases with marked exaggeration of reflex responses, whose examination has not detected any disease.

But what is very important is the agreement and symmetry or asymmetry of the reflexes, that is, the comparison of the response of a reflex with the rest of the responses of the reflexes of the same person; or comparing the response of one side with the response of the same reflex on the opposite side of the body.

Furthermore, the result of the reflex examination will always be evaluated within the results of the rest of the neurological examination before it is considered as an expression of disease.

For a standardized recording, the degree of response of the deep tendon reflexes is evaluated on a scale from 0 to 4 crosses (+).

There is no response (areflexia).

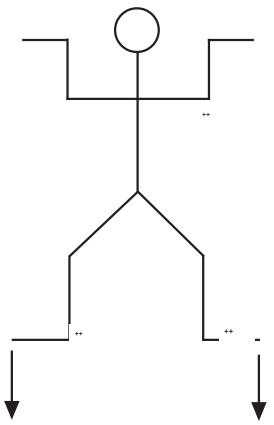
1+ Poor response, decreased (hyporeflexia).

2+ Normal.

3+ Increased (can be normal).

4 + Hyperactive, excited (hyperreflexia).

The responses of the deep tendon reflexes and the cutaneous-plantar reflex can be recorded on a stick figure like this:



The downward arrows indicate that the cutaneous-plantar reflex is normal and the upward arrows indicate that there is a Babinski sign.

SENSITIVITY

CONCEPT

Sensitivity is the ability of the cerebral cortex to react to the stimuli provided to it by the centripetal conductive pathways, with an excitation process that runs parallel to a psychic process. The simplest expression of this function is observed in the simple irritability of single-celled organisms.

Strictly speaking, sensitivity includes the sensations of the sensory nerves, as opposed to the sensations that come from the sense organs (eye, ear, organ of smell, taste) . For diagnostic research, only the sensations perceived by consciousness, about which patients can report, are important.

PRODUCTION MECHANISM

In 1906 Sherrington proposed the following classification for bodily sensations:

Exteroceptive sensitivity. Reports on changes in the environment; example: skin sensitivity.

Proprioceptive sensitivity. It informs about the movements of the body and its position in space; example: tenderness coming from the bones, muscles and joints.

Interoceptive sensitivity. Reports on visceral activity.

Head's (1920) studies introduced terms

As:

Hyperesthesia. Unpleasant increase in skin sensitivity.

Epicritic sensitivity. Finer and more selective skin sensation, which allows differentiating small degrees of heat, and discrimination of tactile sensitivity, such as differentiating distance between two points, or light touches; and differentiate different objects that cause the sensation, such as a pin, cotton or paper.

Protopathic sensitivity. It allows you to appreciate gross skin sensations, such as pain and extreme temperature changes.

Other terms that we should know and that are frequently used in texts that deal with this topic are the following:

Conscious surface sensitivity. It includes the sensations of touch, hot and cold, and pain (tactile, thermal and painful).

Conscious deep sensitivity. It refers to the sensations originating in tendons, muscles, bones, deep dermis or joints, they are: the sense of pressure or barsesthesia (from baros: pressure; aisthesis: sensitivity), which consists of the appreciation of the pressure exerted in different parts of the body; barognosia, recognition of the weight of objects; vibratory sensitivity or palesthesia (from the Greek pallein; to shake), which consists of the perception of vibratory stimuli, such as that of the tuning fork; the sense of segmental attitudes or bathyesthesia, through which the person is able to know the exact position in which the various parts of his body are located, without the help of vision and stereognosia, better called stereognostic perception (from the Greek stereos: solid; gnosis: knowledge) allows the subject to recognize an object through the different types of sensitivity that we have exposed. Thus, without the aid of vision (with touch only) the subject establishes the shape, contour, weight, size and other qualities, being able to recognize them and mention them by name. This type of sensitivity requires the participation of the cerebral cortex, and to explore it it is essential to be certain that the subject does not have the other, more basic forms of sensitivity altered.

Visceral sensitivity. This type of sensitivity is generally painful, like that of the testicle, breast, trachea or eyeball, whose compression awakens pain; but it includes other sensations, such as fullness or emptiness of the stomach, which induces rejection or craving for food; or bladder filling, which induces urination.

There are various types of peripheral receptors, located in the skin, and to which the ability to perceive one or another type of sensitivity is attributed. We can cite the following:

Meissner corpuscles and Merkel disks Krause bulbs	Surface touch Cold
Ruffini corpuscles Free nerve endings.. Pain	Heat

More recently, arguments have been provided against this specificity or specialization of the recipients; but this aspect has no significance in the study of the semiology of sensitivity, so we will not insist on it.

Pathways of sensitivity

The first neuron of the sensory pathway is located in the spinal ganglion of the posterior root of the

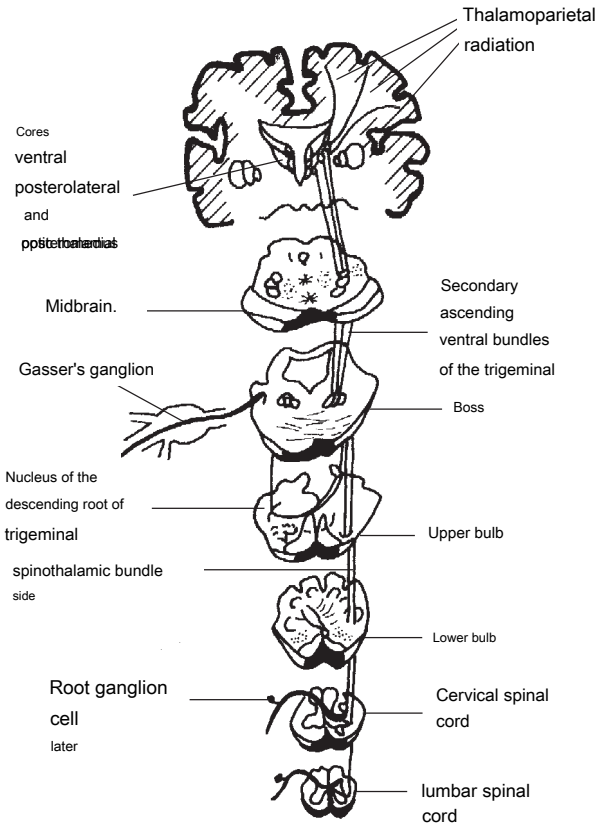


Fig. 14.31 Sensitivity pathways: short fibers (thermal and pain sensitivity).

spinal cord, or in its counterparts of the cranial nerves, such as the Gasserian ganglion, for the sensory branch of the trigeminal nerve. These are neurons that have a peripheral extension, which reaches the cutaneous receptors, and a central extension, which conducts sensations to the spinal cord. These central processes, together, form the posterior spinal root. These posterior root fibers can be subdivided into short, medium and long, according to the distance they travel before synapsing within the spinal cord or medulla oblongata.

The short, unmyelinated or poorly myelinated fibers penetrate through Lissauer's zone and end up synapsing on the neurons of the substantia gelatinosa of Rolando at the end of the posterior horn of the cord, in the same entry segment. It is accepted that these fibers conduct pain and thermal sensitivity.

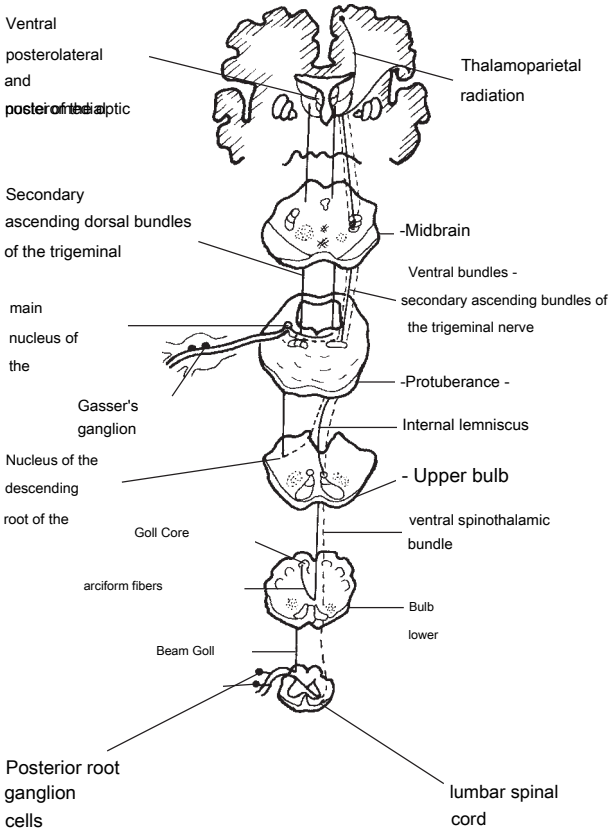
The axons of this second neuron cross to the opposite side through the anterior gray commissure, in their path in the cord they form the lateral spinothalamic bundle, which ascends towards higher centers. Upon reaching the medulla oblongata, this spinothalamic bundle remains located laterally, near the ascending fibers of the sensory branch of the trigeminal nerve. After its ascent through the brain stem, it ends in the anteroposterolateral nucleus of the thalamus. From this third neuron the pathway that establishes the connection with the cerebral cortex, in the parietal gyrus, passing first through the posterior arm of the internal capsule (fig. 14.31).

The medium fibers conduct tactile sensitivity.

They travel through several floors or spinal segments before synapsing with neurons in the posterior horn of the spinal cord. The axons of these neurons cross the midline through the gray commissure or the white commissure, forming the anterior spinothalamic bundle in the spinal half opposite to its entrance. Like the previous one, the medullary spinothalamic tract ascends through the spinal cord and brain stem, ending in the posterolateroanterior nucleus of the thalamus. Thalamic neurons, like other specific sensory connections, send their axons through the internal capsule (posterior arm) to the sensory area of the cerebral cortex, postcentral gyrus (ascending parietal gyrus). As it ascends the brain stem, the anterior spinothalamic fasciculus joins the medial lemniscus or medial ribbon of Reil (Fig. 14.32).

The long fibers ascend through the posterior cord of the medulla without synapsing in the posterior horn or crossing to the opposite side, forming the fascicles of Goll and Burdach (gracilis and cuneiform) to finally synapse in the gracilis and cuneiform nuclei of the medulla oblongata. These fibers conduct sensation

Fig. 14.32 Sensitivity pathways: medium fibers (tactile sensitivity).



deep conscious and discriminatory tactile sensitivity. The second neuron of this pathway (located in the gracilis and cuneiform nuclei) sends its axons to the opposite side of the medulla oblongata and forming the medial lemniscus or medial ribbon of Reil, they ascend to the thalamus, also ending in the posterolateroanterior nucleus.
From here on, its connection with the cerebral cortex is similar to that described for other forms of sensitivity (fig. 14.33).
Another group of these long fibers establishes synapses in a sector of the posterior horn (Clarke's columns and Stilling's nuclei); The fibers of this second neuron form the direct spinocerebellar tract (Fleching's tract) and the crossed spinocerebellar tract (Gowers' tract); Both are located in the lateral cord and when they reach the medulla oblongata and the pons, they penetrate the cerebellum through the cerebellar peduncles, ending in the region of the vermis. They guide deep unconscious sensitivity and are responsible for the deep information (muscle, bones, joint) necessary to maintain the body's attitude, balance and coordination.

The medial lemniscus is located, after its crossing (sensory decussation or decussation of the medial lemniscus) behind the pyramidal fasciculus. Both junctions, the sensory and motor, are located more or less at the same level of the medulla oblongata, the sensory, slightly higher than the pyramidal decussation.

As can be understood from this description, the nuclei of Goll (gracilis) and Burdach (cuneiform) are nothing more than extensions in the medulla oblongata of the posterior horns of the medulla.

It can also be deduced that the sensitivity pathway has at least three neurons: the first, located in the spinal ganglion, the second, which has its body in the posterior horn of the spinal cord or in equivalent nuclei of the brain stem. The axons of this second neuron cross to the opposite side at some point during their journey to the thalamus. The third neuron is in the thalamus and its axons terminate in the cerebral cortex. It is also clear that tactile and deep conscious sensitivity follow a common pathway from the brain stem (medial lemniscus);
while the thermoalgesic sensitivity remains separate (in the lateral spinothalamic bundle) until it reaches the optic thalamus.

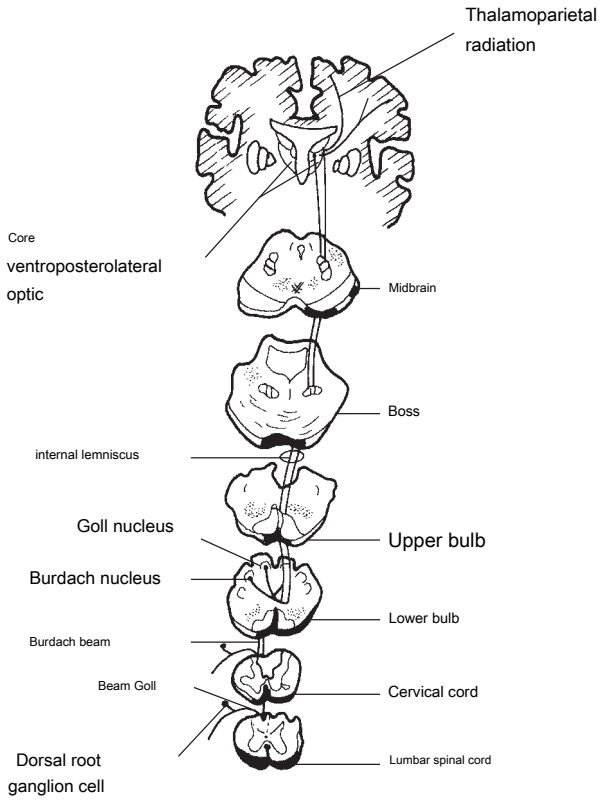
As the crossing of the pathways of superficial sensitivity takes place at the level of the spinal cord, while that of the pathways of deep sensitivity occurs in the medulla oblongata, it is understood that a left spinal hemisection will interest the pathways of thermoalgesic and gross tactile sensitivity that come from the right side of the body (already crossed) and those of deep and epicritic tactile sensitivity that come from the left. (not yet crossed), so there will be deep anesthesia.

gives on the side of the injury and superficial anesthesia on the side opposite the injury. This is what happens in the so-called Brown-Sequard syndrome. The pathways of tactile and thermoalgesic sensitivities that we have just described constitute the so-called lemniscal pathway or lemniscal system with few synapses and that more or less directly reaches the cortical sensory centers. This system has a fast conduction speed, 90 m/s, and conducts specific sensory stimuli with great precision that end up projecting onto areas SI and SII (retrolandic ascending parietal area and parietorlandic edges of the Sylvian valley, respectively).

Now, along the reticular substance of the brain stem, there is an ascending projection system whose function is related to the state of waking consciousness, sleep and comas. This system is multineuronal, polysynaptic and is formed and enriched by collaterals that are given to it by the ascending sensory systems (Barraquer-Bordás). In this way, fibers from the thermal and pain sensory pathways, and from the lateral lemniscus (acoustic pathway) are integrated into it.

The impulses that originate from the lemniscal system exit through two poles: the lower pole that supplies descending inhibitory or facilitatory pathways, which project

Fig. 14.33 Proprioceptive sensitivity pathways: long fibers (conscious deep sensitivity and discriminative tactile sensitivity).



so on the interneurons and motoneurons of the spinal cord regulating their activity; the upper pole, from which diffuse ascending pathways originate that play a fundamental role in maintaining the state of wakefulness and in the level of activity of the integration structures of the brain (ascending reticular activating system: RAAS).

This authentic engine of the nervous system bases its energy on the contribution of chemical factors, which act humorally, especially adrenaline and CO, and physical factors, contributed by sensory, motor and vegetative discharges. The impulses from these different origins lose their specific character when they merge into integrating neurons, becoming solely energetic factors, more or less powerful, depending on their nature and intensity.

The reticular substance thus creates a harmonious correlation between the factors of relational life and those of vegetative life, constituting an authentic final common pathway for the set of information from the internal and external media. By functioning as a whole, it will adjust the activity of the set of nervous and endocrine functions that it presides over, to maintain life.

This system is part of the extralemniscal system, and is characterized by being non-specific, and also having its representation in thalamic nuclei, and like the system lemniscal ma projects to the cerebral cortex, but not to specific areas, but to the entire cortex. Its activation in the sleeping animal causes it to wake up ("awakening reaction"). Damage to this system, on the contrary, produces a coma-like state.

In this way, sensitivity can reach the cerebral cortex through these two systems, producing the activation of the ascending reticular activating system, not a specific perception, but a state of consciousness or alertness; while the lemniscal system would allow us to perceive the specific sensations that stimulate it.

Pathways of visceral sensitivity. They differ, in part, from the previous ones. The stimuli that awaken visceral sensitivity follow the sympathetic and parasympaic nerves, reach the laterovertebral or cranial sympathetic ganglia, and then, through the rami communicans, reach the posterior root of the spinal nerves and Clarke's column of the spinal cord; From now on they continue along the paths of general sensitivity.

Cortical sensory centers. The sensory centers are found in the cortex of the parietal gyri and, in particular, the postcentral gyrus (ascending parietal gyrus) and in the foot of the first and second parietal gyri. These regions constitute the somesthetic area of the cortex where the superficial and deep sensations of the opposite half are collected.

ta of the body. The sensory center of the face occupies the lower part of the postcentral gyrus (ascending parietal gyrus) ; the center of the upper limb, the middle part of the ascending parietal and the center of the lower limb, the upper part of the ascending parietal and the paracentral lobe. In summary, the cortical sensory centers have the same arrangement as the cortical motor centers, from which they are separated only by the central sulcus (Roland's fissure). There is much debate about the functioning of these cortical centers and the role they play in the appreciation of sensations. Head has maintained that the cortical somesthetic area corresponds to discriminative power and stereognosia.

A subject who has a cortical lesion of the sensory area will be able to perceive the corresponding sensation, but will have difficulty judging and discriminating the sensation and will be unable to synthesize the different sensations that allow the recognition of an object. Many sensory excitations do not reach the cortex; they would become conscious in the thalamus like pain. The thalamus forms a very important subcortical sensory center, which ensures protopathic sensitivity and confers the affective tone, of pleasure or pain, that is associated with certain sensations. Hence, when the thalamus is freed from cortical control, it is observed that certain sensations are intensely felt (hyperpathy).

Root and peripheral distribution of sensitivity.

As in the case of motility, peripheral sensory territories can be distinguished that correspond to the sensory distribution area of a spinal nerve and radicular sensory distribution territories that correspond to the distribution area of each posterior sensory root.

This is because the peripheral nerve receives sensory fibers that come from the various branches that have formed the plexuses (from which the nerves come) and also because the same root participates in the formation of several spinal nerves. The root sensory topography is, therefore, different from the peripheral sensory topography. Thus, for example, the section of the ulnar nerve determines the loss of sensitivity in the ulnar edge of the hand;

To achieve an equal injury, it will be necessary to section the eighth cervical root and the first dorsal root, since the ulnar root comes from these two roots, but since these two roots simultaneously give rise to the internal brachialcutaneous root, the loss of sensitivity will not be limited to the ulnar edge of the hand, but will extend to the inner edge of the forearm and arm.

In summary, sensory topography includes two types: peripheral and radicular. The peripheral one coincides with the distribution area of the peripheral nerve, while the radicular one is made up of the extremities.

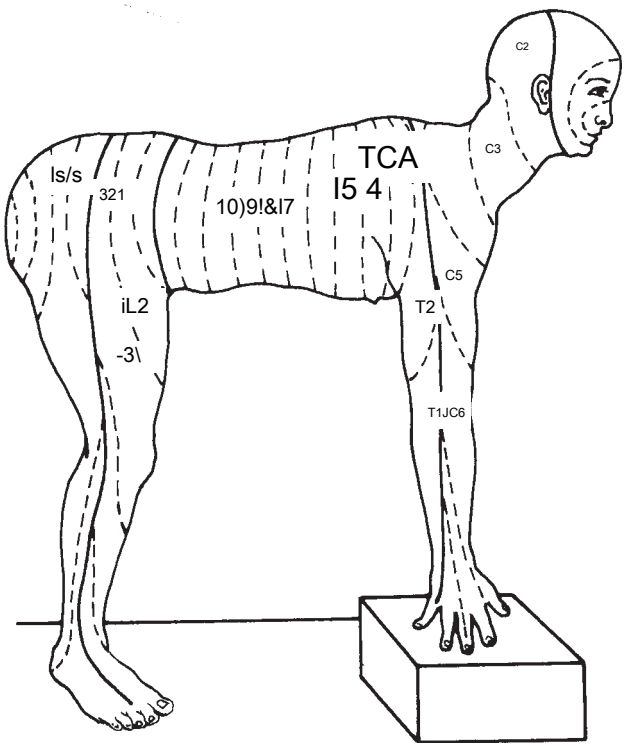


Fig. 14.34 Segmental or radicular distribution of sensitivity.

by bands perpendicular to it (fig. 14.34). It should be noted that the bands are not strictly separated, but rather partially overlap each other. These Zones have been able to be determined, thanks to Head's studies on herpes zoster, which is an inflammation of the posterior root ganglion, due to a specific virus.

EXPLORATION TECHNIQUES

From the semiological point of view, it is useful to distinguish subjective sensitivity from objective sensitivity, subjective sensitivity being understood as those sensitive manifestations that the subject spontaneously observes; unlike objective sensitivity, which corresponds to the sensory manifestations that the clinician can highlight through various excitations that he uses following a specific technique. In reality, sensitivity is always subjective and this division is arbitrary, but it serves to distinguish what the examiner discovers through provocative procedures, from what the examinee spontaneously accuses. Objective sensitivity is explored according to a special technique for each of its forms.

In general, the conditions that require a good objective sensitivity test are:

1. Require good attention from the subject being explored, previously explaining how
   You should respond as soon as you experience the sensation following the application of the stimulus.
2. The subject must not see what the examiner does when examining him. To do this, he is made to close his eyes or his vision is covered with a handkerchief, or he is ordered to look at the opposite side to the one being examined.
3. The explorer should keep in mind that the study of sensitivity quickly tires the person, and that, therefore, a thorough study of it sometimes requires several sessions.

Exploration of surface sensitivity

These explorations must be done in accordance with the descriptions made when talking about the metamers, and for this we recommend that the study be done following lines perpendicular to the axes or bands of the dermatomes, in such a way that when passing through one of them (if it is altered), the subject notices the difference" with the others. Once an " altered band" is detected, we explore it in a second time, in its entire extension. In the limbs we will do it in a circular manner to them: and on the thorax, abdomen and back, vertically or perpendicular to the lines of the dermatome.

1. Tactile sensitivity. To do this, use a piece of cotton, a brush, or your fingertips. Different points of the skin and also of the mucous membranes such as the nose, mouth, etc., if necessary, will be successively touched with one of these elements. Avoid putting pressure on the excited points; the excitation must be simple contact. It will be touched quickly two or three times in a row and the subject will be asked how many times it has been stimulated. The Weber compass can also be used with its graduated aperture, which allows the so-called Weber circles to be investigated, that is, the minimum distance at which the simultaneous contact between two points on the skin is appreciated by the subject, as two different tactile sensations.

2. Painful sensitivity. It is explored using the tip of a pin or a needle or an algesiometer. The technique is similar to that used for exploring tactile sensitivity. Practically, both sensitivities, tactile and painful, can be investigated, using a piece of cotton and a common injection needle and instructing the person to answer: "it touches me" or "it pricks me" depending on the sensation they experience.
   mind.
3. Thermal sensitivity. To explore sensitivity to cold and heat, two test tubes are used, one containing very hot water and the other cold water or ice chips. If it is not possible to obtain these means, a heated instrument can be used. Care must be taken with the hot tube so that it does not burn.

for too long in contact with the skin, to avoid burns.

When studying surface sensitivity, it will be advisable to compare symmetrical points and repeat the exploration several times, to be sure of the existence of disturbances; Furthermore, efforts will be made not to apply the stimuli very immediately, one after the other, to avoid confusion in the responses.

If sensitivity disorders are found, an attempt will be made to mark the limits of the altered areas, drawing lines on the skin with a dermographic pencil that correspond to the affected regions. In this way, the height, extent and distribution of existing disorders can be established.

Exploration of deep sensitivity

1. Exploration of pressure sensitivity (baresthesia) and weight appreciation (barognosia). To explore pressure sensitivity, tactile and thermal sensitivities must be avoided. For common clinical use, it is sufficient to press on different points of the body, with the tip of a finger, generally the index, and ask the subject which point has been pressed the most. When a more delicate examination of barsesthesia is required, metal discs of different weights are used to exert pressure or special instruments (such as the Eulemburg barsesthesiometer).

Barognosia is explored through objects of similar shape and of different weights, for example, weights of different values that are placed on the subject's hand. Normally, a subject should notice an increase or difference of one third in the weight of two different objects.

2. Exploration of vibratory sensitivity (palesthesia).

Palesthesia is studied with the help of a tuning fork, with 128 vibrations per second, which is made to vibrate by hitting its "U" branch, and which is immediately applied by the foot on a bone surface, the epiphysis of the long bones, the tibia, for example.

The person, who keeps his eyes closed, perceives a sensation of trepidation or vibration on the bone, while the tuning fork vibrates, which he generally compares to electricity. The explorer (to know the state of paleesthesia) asks the subject what sensation

sation has.

3. Exploration of the meaning of segmental attitudes (bathyesthesia). The exploration of bathyesthesia is practically equivalent to studying joint and muscle sensitivity. The procedure proceeds without the subject looking at what the explorer is going to do; any joint is moved passively, in different directions, and it is

It stops in a certain position, and is then asked in what position it has been placed, or it is instructed to actively reproduce this position with the joint on the opposite side (naturally without looking). Typically, you use your fingers or toe, thumb or big toe, for example, and ask how the finger is, whether together or apart, up or down. (The technical terms "in flexion", "in adduction", "abduction" should not be used with the subject because he does not understand them.) During this examination, the muscles of the joints examined must be completely relaxed. If a thorough examination is desired, the various segments of a limb are studied, starting, for example, with the interphalangeal joints and then with the metacarpophalangeal joints, the wrist, the elbow, etc. (if it is the upper limb).

4. Exploration of deep painful sensitivity.

The sensitivity of muscles and tendons to deep compression is known as deep painful sensitivity. Normally, moderate compression of the muscles and tendons is painless, but in certain pathological states (polyneuritis, myositis, etc.) the muscles are sensitive to the slightest pressure, or on the contrary, in others (tabes), the most vigorous compression does not cause pain.

The examination is simple: it consists of compressing, with the hand, the muscle masses or pinching the accessible tendons, such as the Achilles tendon, for example.

5. Exploration of stereognosis. It is explored in the following way: without the subject looking, common objects (a coin, a key, a pencil, a pin, etc.) are placed in the palm of their hand, and they are invited to study them, moving them between their fingers; Next, you must say what its characteristics are: shape, size, consistency, etc., and then name them. Recognizing the aforementioned qualities of the object, that is, making the primary identification, constitutes the stereognostic meaning. Naming it, that is, its secondary identification, already means the intervention of factors of the cerebral cortex.

If the subject has a motor disorder, for example, hemiplegia, the examiner will slide the object through his or her hand, keeping it passively closed.

In order to explore stereognosia, the other forms of superficial and deep sensitivity already described must be preserved.

Exploration of visceral sensitivity

The examination of visceral sensitivity is carried out by compressing the testicle, trachea,

epigastrium, or breast. The sensitivity of the bladder is explored by distending it by probing and injecting fluid inside. It is enough to introduce 120-150 mL of liquid, in the normal subject, to provoke the desire to urinate. This examination is of exceptional use in clinical settings, and has the risk of causing urinary tract infection. The sensitivity of other viscera escapes ordinary exploration, except in certain pathological circumstances.

GUIDE AND RECORD OF THE EXPLORATION OF THE NERVOUS SYSTEM

1. Facies
2. Standing attitude
3. Attitude in bed 4. Gait

ì No characteristics of the neurological process

i (already explored in the î general physical examination)

5. Consciousness: fully awake, alert, obtunded, stuporous, comatose.
6. Orientation in time, space and person: oriented or not.
7. Memory: immediate, recent (anterograde) and distant or distant (retrograde).
8. Language: clear and precise, clumsy, dyslalic, dysarthric, brady or tachylalic, aphasia).
9. Taxia (coordination):

Simple Romberg Static Coordination
i Sensitized Romberg i
Bárány index Index-index Dynamic coordination index-nose Heel-knee î Diadochokinesia

10. Praxia: transitive, intransitive and imitative movements.

11. Motility:

Volunteer i
- active movements
  - 1 Activates Segmental muscle strength
    - Barré and Mingazzini maneuvers
- Yo
- Yo muscle tone
- i Passive
  - Meningeal signs: neck stiffness; Kernig and Brudzinski maneuvers

Involuntary: does not exist (or tics, tremors, convulsions, chorea, athetosis, ballismus, fasciculations, fibrillations).

12. Trophism: (explored in skin and osteomyo-articular system, SOMA).

13. Reflectivity:

Osteotendinous
- i Superciliary
- Nasopalpebral
- i Mental
- i Tricipital
- Bicipital
- # Styleradial
- i Ulnapronator
- i Patellar or patellar
- Aquiliano
- 1 Midplantar

Cutaneomucosa
- i Conjunctival
- i Corneal
- i Sneezing
- i Nauseous
- upper cutaneousabdominals,
- i middle and lower
- Cremasterian
- and Cutaneoplantar

14. Sensitivity:

Superficial: tactile, thermal, painful.

Baresthesia
- i Barognosia (weight sense) (
- Deep pressure sense) (
- Bathyesthesia position sense) (
- i vibration sense) (
- Palesthesia association sense)

NERVOUS SYSTEM PHYSICAL EXPLORATION OF THE CRANIAL PAIRS

15

General considerations

The cranial nerves, commonly called cranial pairs, have classically been considered to consist of twelve pairs of nerve trunks, which have their apparent origins on the brain surface and, after more or less long journeys inside the cranial cavity, they leave it through orifices located in the cranial base to reach their areas of innervation.

The examination of the cranial nerves is essential in the study of the nervous system. The proper localization of lesions that affect the cranial nerves requires: skill in the examination and knowledge of the neuroanatomy of the brain stem and cranial nerves.

Although these pairs receive particular names, there is a tendency to designate them by Roman numerals that are assigned based on their cephalocaudal order of brain implantation. Except for the I or olfactory pair and the II or optic pair (which are not true nerves, but extensions of the brain, although they are still considered among the cranial nerves), to which we add a part of the XI or accessory pair that arises from the high cervical cord, all the remaining cranial nerves have their apparent origin along the brain stem.

The apparent origin of a cranial nerve is that area of the brain surface in which said nerve is implanted or fixed. The motor fibers of the cranial nerves have their real origin in accumulations of neurons (masses of gray matter that form the motor nuclei) located deep in the brain, from which axons originate that, forming their respective nerves, conduct nervous impulses towards the effectors (muscles or glands).

The afferent or sensory fibers of these nerves have their real origins in accumulations of neurons located outside the brain, in the so-called afferent cranial ganglia; Each cranial nerve with afferent components has one or more specific ganglia.

The neurons of these afferent cranial ganglia have extensions that originate from receptors located in the periphery; In turn, other extensions originate from these ganglion neurons that, reaching the neuraxis, synapse in other neuronal accumulations included in the brain, which constitute the sensory nuclei of the cranial nerves.

Depending on whether these nerves contain afferent fibers, motor fibers, or both types, cranial nerves can be sensory,

engines or mixed, respectively. The sensory nerves will have afferent cranial ganglia and sensory nuclei; Motor nerves will have only motor nuclei and mixed nerves will have afferent cranial ganglia and sensory and motor nuclei. Both afferent and efferent pairs can be somatic or visceral, taking into account their destination. Visceral fibers can, in turn, belong to the sympathetic or parasympathetic system.

According to these considerations, the cranial nerves are classified as:

1. Sensory and afferent pairs. The olfactory (I pair), the optic (II pair) and the vestibulocochlear or statoacoustic (VIII pair). Let us remember that pairs I and II are not true nerves, hence their characteristics deviate from the general considerations that we are exposing.

2. Motor or efferent pairs. The common ocular motor or Oculomotor (III pair), the pathetic or trochlear (IV pair), the external ocular motor, abductor or abducens (VI pair), the spinal or accessory (XI pair) and the hypoglossal (XII pair).
3. Mixed pairs. The trigeminal (V pair), the facial (VII pair), the glossopharyngeal (IX pair) and the vagus or pneumogastric (X pair).

The nuclei of the cranial nerves, in general, are located higher in the neuroaxis the lower the order number corresponding to the cranial nerve we are considering. Thus, for example, the nuclei corresponding to the II and IV pairs are in the cerebral peduncles (mesencephalon), those of the V and VII pairs in the pons or pons and in the medulla oblongata or medulla oblongata, while those of the VI and VIII pairs are only pontine or pontine; Finally, those of the IX, X, XI and XII pairs are located in the medulla oblongata.

It is very important to know that these nuclei are located on the same side of the neuroaxis, right or left, to which the cranial nerve corresponds or has its apparent origin; For example, the nuclei corresponding to the facial on the right side, which innervates structures of the right hemiface, are located on the right side of the pons. The IV nerve is the only one that crosses after its emergence from the brain stem, to innervate the side opposite to its nucleus of origin.

All cranial motor nuclei, from which nerve fibers destined for voluntary striated musculature (somatic motor nuclei) originate, receive corticonuclear fibers from the opposite cerebral hemisphere, coming from the corticonuclear motor area located in the juxtacisural part of the precentral gyrus or ascending frontal gyrus. The neurons from which the axons emerge that will integrate that corticonuclear pathway or bundle geniculate, are located in the lowest part of the precentral gyrus, at a lower level the higher the order number of the cranial nerve. Let us remember that in the cortex of this gyrus the location of the corticonuclear neurons corresponding to the voluntary striated muscles of the different parts of the body reproduce the caricature of a subject with the head downwards and the feet towards the interhemispheric fissure (fig. 15.1).

But the remarkable thing about these pyramidal fibers that join the cerebral cortex to the somaic motor nuclei is that they come from the opposite cerebral hemisphere, as we have already said, their decussation being verified little by little at different heights of the brain stem, at the same approximate level at which the motor nucleus for which they are intended is located.

On the other hand, all somatic motor nuclei receive some pyramidal fibers from the cortex on the same side (with the exception of a part of the facial nucleus). Therefore, the vast majority of somatic cranial motor nuclei receive bilateral connections.

The apparent origins of the cranial nerves can be seen in Figure 15.2.

Since their emergence from the neuraxis, the cranial nerves have an intracranial path that varies in length, in which they are more or less close to each other, to finally exit the cranial cavity through specific holes that we find at the base of the skull. From a semiological point of view, it is essential to know these paths, since in many cases they will constitute an important orientation to locate the site of the lesion in which several cranial nerves are simultaneously affected, thus creating different syndromes. For example, in the cavernous sinus, the III and IV pairs, the ophthalmic branch of the V and VI cranial nerves, are located very close to each other, as can be seen in Figure 15.3.

It is also important to know the so-called "cerebellar pontine angle", a place where the VII and VIII pairs are practically attached, and very close to them the V and VI pairs. Therefore, in tumors of the cerebellopontine angle these nerves are compressed, which is expressed by the characteristic symptoms (Fig. 15.4).

The usefulness of knowing through which bone opening the cranial nerves leave the cranial cavity will be understood when we consider that in each of these openings, located at the cranial base, several nerves of different origin and path can coincide. In figure 15.5 these holes can be seen, indicating the pairs that come out of each of them.

Specifically, we will say that:

a) Olfactory nerve. The processes of the olfactory cells of the nasal mucosa are sieved through the cribriform plate of the ethmoid.

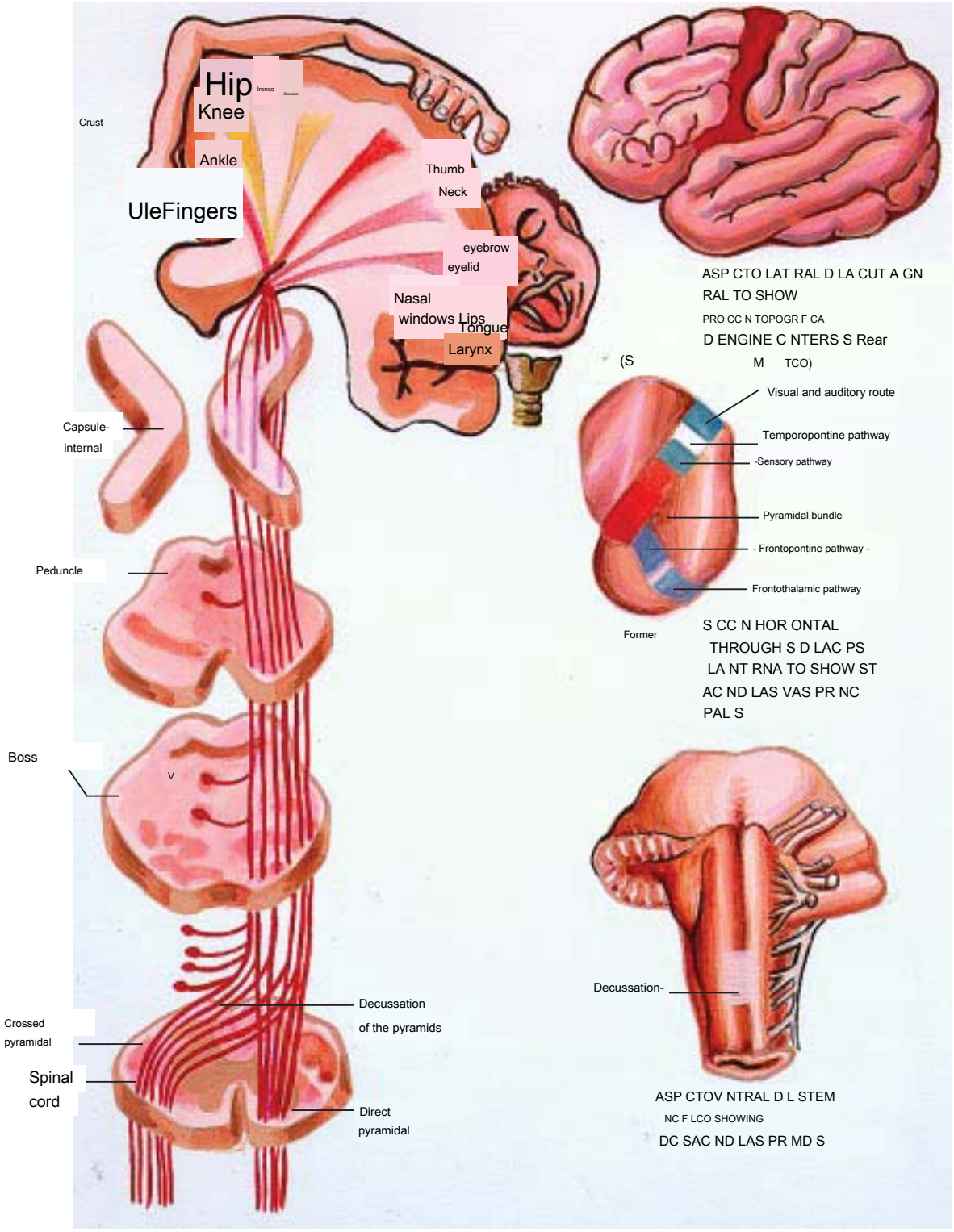


Fig. 15.1 Representation of voluntary movements in the cerebral cortex.

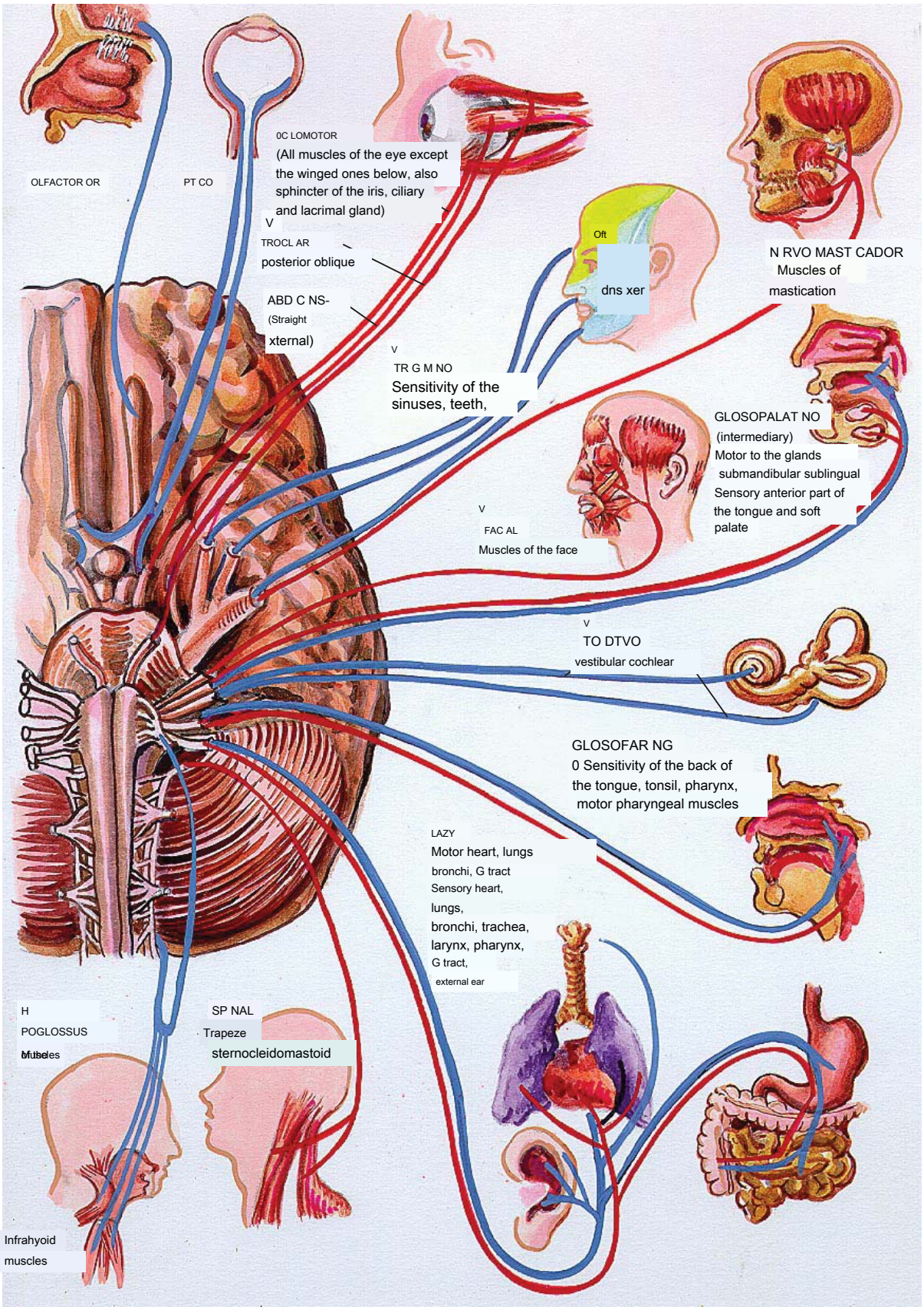


Fig. 15.2 Apparent origin of the cranial nerves. Base of the brain.

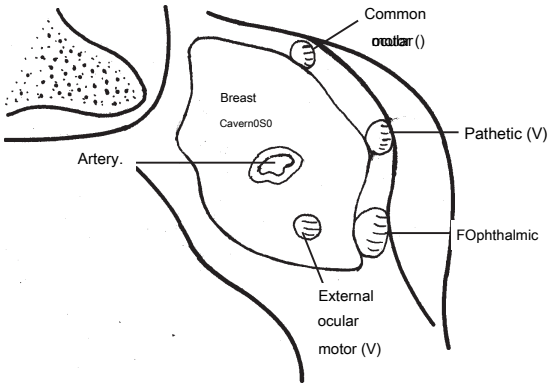


Fig. 15.3 Distribution of the III, IV and VI pairs in the sinus ca-
verous

b) Optic nerve. It passes through the optic foramen along with the ophthalmic artery.

c) Common ocular motor, trochlear and abductor. They reach the respective orbit through the superior orbital fissure or sphenoidal fissure on the corresponding side.

d) Trigeminal. The ophthalmic branch exits through the superior orbital fissure or sphenoidal fissure; the maxillary branch, through the foramen rotundum, and the mandibular branch, through the foramen ovale.

e) Facial and auditory. They pass through the ear canal
terno

f) Glossopharyngeal, vagus or pneumogastric and accessory or spinal. They exit the skull through the jugular foramen or posterior tear, very close to the point where the transverse sinus is continuous with the gulf of the internal jugular.
na.

g) Hypoglossus. It passes through the anterior condylar foramen.

Next we will do a particular study of the cranial nerves, which are:

- Olfactory nerve: I pair.
- Optic nerve: II pair.
- Common oculomotor, pathetic (trochlear) and external ocular motor (abducens) nerves: II, IV and VI pairs.
- Trigeminal nerve: V pair.
- Facial nerve: VII pair.
- Statoacoustic nerve: VIII pair.
- Glossopharyngeal, pneumogastric (vagus) and accessory nerves: IX, X and XI pairs.
- Hypoglossal nerve: XII pair.

IPAR OLFACTORY NERVE

From the histological, ontogenetic, phylogenetic and functional points of view, this pair is not a true nerve, we insist, but rather a telencephalic extension that is part of the so-called visceral brain or rhinencephalon.

ANATOMY AND PHYSIOLOGY FIG.

From the bipolar olfactory neurons (1st olfactory or Schultze cells) included in the nasal mucosa that covers the superior and middle turbinates, as well as the upper part of the nasal septum, and which present very short peripheral extensions that reach the free surface of the mucosa, unmyelinated axons arise that, following an ascending path, cross the cribriform plate of the ethmoid to make synapses with neurons of the medulla. olfactory (mitral cells), which are found resting on the respective side of the horizontal plate of the ethmoid. The axons of the neurons of the olfactory bulb form the so-called olfactory band, with an anteroposterior course, which, upon reaching the anterior limits of the anterior perforated substance, divides into the so-called medial and lateral olfactory striae. The axons of these striae will synapse directly or indirectly with neurons of the rhinencephalic complex, especially the uncus region of the hippocampus. Among the rhinencephalic structures with which the olfactory pathway establishes a relationship, we mention the hippocampus, the amygdaloid nucleus, the piriformis area, the thalamus, the mammillary bodies, the habenular nucleus, the trigone and others. In this way, reflex connections are established with the nuclei of the other cranial and spinal nerves that have functional activity in swallowing and digestion.

The peripheral endings of the Schultze cells are chemically stimulated by odorous particles, which are suspended in the air circulating through the nasal passages.

EXPLORATION TECHNIQUES

1. Small bottles should be prepared with substances with known, common or common odors, and that are not irritating. Among them the smell of cloves, coffee, soap, perfume, turpentine, camphor, etc. Ammonia, vinegar, formalin or other substances should not be used, which would irritate the sensitive endings of the V pair.

2. Ask the person to: occlude one nostril with their finger, keep their mouth closed, and close their eyes.
3. Present several familiar odors, placing the mouth of the container containing the substance being used, under the nostril being examined.
4. Each side of the nostrils should be explored separately. Repeat the procedure in the other nostril.

5. We must first ask if he or she smells the smell and if he answers positively, he is urged to identify the smell.

6. Carefully write down, indicating for each nostril, what the result of the test is.

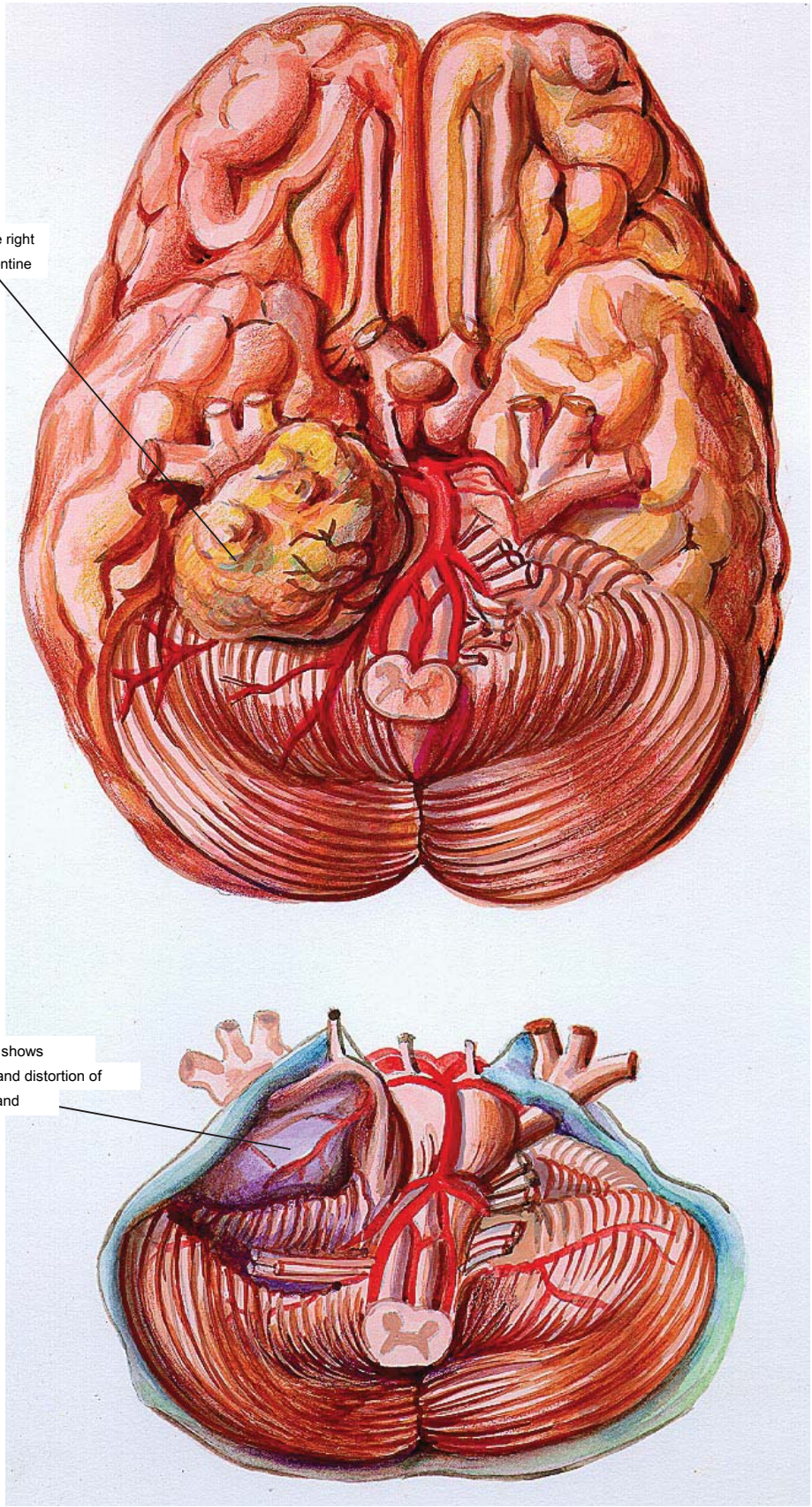


Fig. 15.4 Tumor of the cerebellopontine angle.

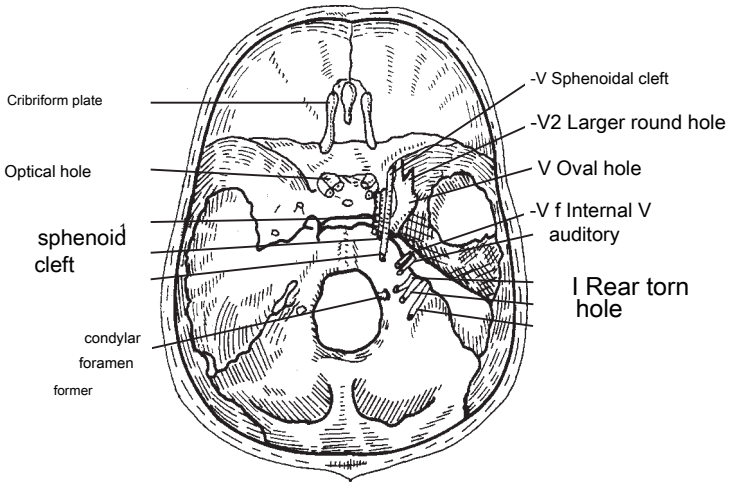


Fig. 15.5 Orifices at the base of the skull and cranial nerves that exit through them: I (olfactory) exits through the cribriform plate; the I| (optical), through the optic hole; pairs III, IV, V" and VI (common oculomotor, pathetic, ophthalmic branch of the trigeminal and external oculomotor), through the sphenoidal cleft; V2 (upper jaw, branch of the trigeminal), through the foramen teres major; V° (inferior jaw, branch of the trigeminal), through the foramen ovale; pairs VII (facial) and VIII (auditory), through the internal auditory canal; pairs I (glossopharyngeal), (pneumogastric) and I (spinal), through the posterior lacerated foramen, and the I| (hypoglossus), through the hole previous condiile0.

7. Before considering a test as positive, one must first make sure that the subject does not have a nasal cold or another condition of the nasal passages that prevents or alters the circulation of air through them.
8. It is important to clarify that although it is not exactly identified, the subject's appreciation of an odor is sufficient to exclude anosmia (loss of smell).

OPTICAL NERVE I| PAIR

Structurally, it represents a bundle of nerve fibers in the brain.

ANATOMY AND PHYSIOLOGY

The peripheral neurons of the optic pathway are the bipolar neurons located in the middle part of the retina, one of whose extensions, the peripheral one, collects the nervous impulses carried by the visual receptors (cones and rods), which, located in the external part of the retina, are impressed by light rays; The central extension of these bipolar neurons synapses with the ganglion cells of the inner part of the retina. From these ganglion cells, the second neuron of the visual pathway, fibers originate from almost the entire retinal surface that will converge at the level of the optic papilla, to leave the eyeball and form the corresponding optic nerve.

The macula is the site of greatest visual acuity, while at the emergence of the optic nerve (the papilla), as there are no cones or rods, there is a small blind spot in the visual field at that level. The optic nerves reach the cranial cavity through the respective optic foramina, they are lost in the optic chiasm located at the level of the floor of the third ventricle, immediately through above the pituitary gland. At the optic chiasm, fibers of the optic nerves from the nasal half of each retina cross with those from the opposite side, while fibers from the temporal half of each retina do not cross. After the optic pathway crosses the chiasm, they constitute the so-called optic ribbons that, according to the partial decussation of the fibers that occurred in the chiasm, each one will be made up of the fibers that come from the temporal half of the retina on the same side and those from the nasal half of the retina on the opposite side (fig. 15.7).

The retinal fibers that will form each optical stripe will end up:

1. Ones in the lateral geniculate body of the thalamus on the same side, which is an accessory thalamic nucleus.
2. Others, continuing along the superior quadrigeminal arm or arm of the superior colliculus that joins the lateral geniculate body to the respective superior quadrigeminal tubercle, end in this tubercle or colliculus.
3. Others, following the path of the superior quadrigeminal arm, border the superior colliculus or the superior colliculus and end up in the pretectal region (located between the quadrigeminal plate and the epithalamus).

The retinal fibers that end in the corresponding lateral geniculate body synapse with neurons therein; The axons of these neurons, ascending through the sublenticular portion of the internal capsule on the same side, with the name of optic radiations or geniculocalcarine bundle, will end in the lips of the calcarine fissure of the occipital lobe, which constitutes the cortical area of vision. The impulses corresponding to the lower halves of the retinas reach the lower lip of the calcarine fissure; to the upper lip, those of the upper retinal halves. The upper fibers run directly posteriorly near

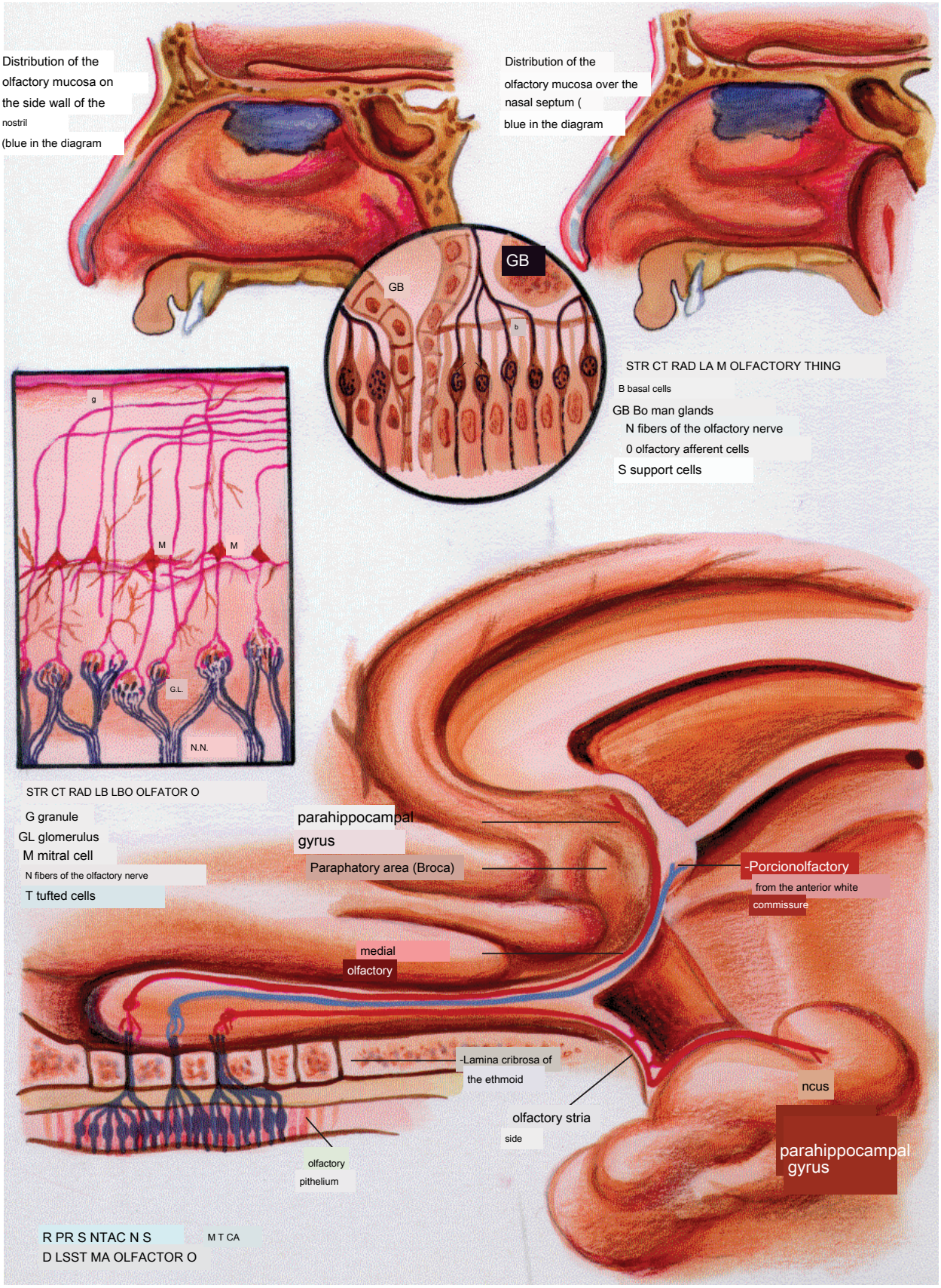


Fig. 15.6 Olfactory nerve: origin, course and termination.

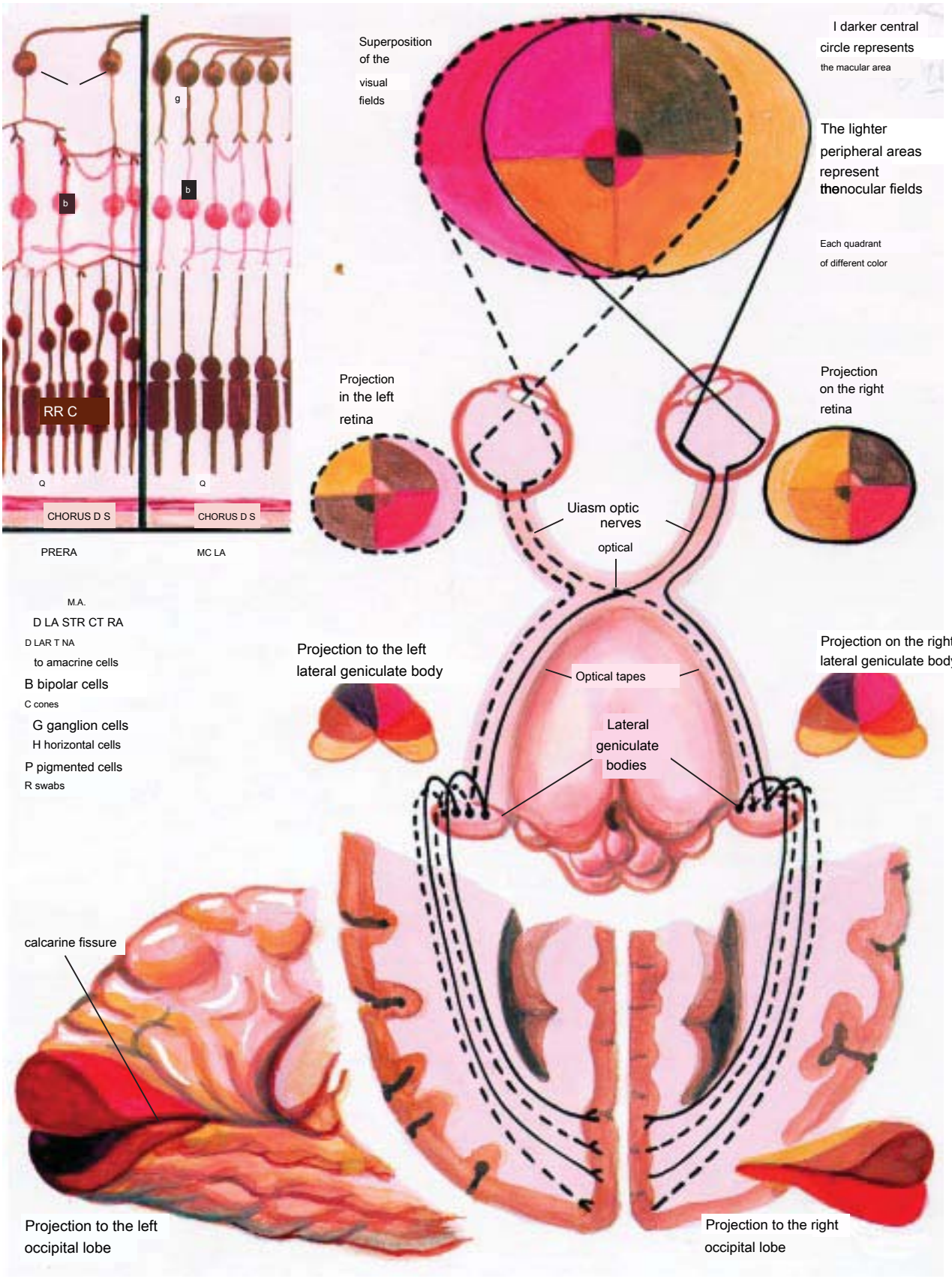


Fig. 15.7 Optic nerve: origin, course and termination.

the external wall of the lateral ventricle. The lower ones deviate forward, forming a handle, before joining the posterior ones and ending in the occipital lobe. The path traced up to here is the one corresponding to the conscious visual pathway.

Pupil size is influenced by many factors.

The afferent pathway for pupillary constriction due to light stimulation (photomotor reflex) travels through the retina, optic nerves, optic tracts, arm of the superior colliculus and reaches the nucleus of the II nerve, where the efferent pathway begins. As a result of semi-decussation of fibers, both in the chiasm and the superior colliculus, both pupils respond to the stimulus. This is the so-called consensual reflection.

The pupils also constrict upon accommodation, the pathways of this reflex are not well defined but it is assumed that they follow a different pathway than the photomotor reflex, probably with cortical connections. Through this reflex, when approaching an object that was distant from us, three processes occur:

1. Bilateral pupillary constriction (due to contraction of the sphincters of the pupils).
2. Convergence of the optical axes (due to contraction of both internal straight lines).
3. Increase in the anteroposterior diameter of the lenses (due to contraction of the ciliary muscles).

Although photomotor, consensual and accommodation reflexes are usually explored in pairs III, IV and VI, we understand that it has been convenient to expose their reflex arcs in this place.

EXPLORATION TECHNIQUES

Examination of the optic nerve includes four different aspects:

1. Visual acuity. From far and near.
2. Perimetry and campimetry.
3. Color vision.
4. Fundus examination.

Visual acuity

The examination of visual acuity includes the evaluation of vision: distance and near.

To determine distance vision, the Snellen chart is used and for near vision, the Jaeger chart is used, which can be replaced by the printed page of a newspaper or telephone directory. When examining near or distance vision in illiterate people or in young children who cannot read, appropriate substitutions are made on the reading signs, replacing letters with figures (Fig. 15.8).

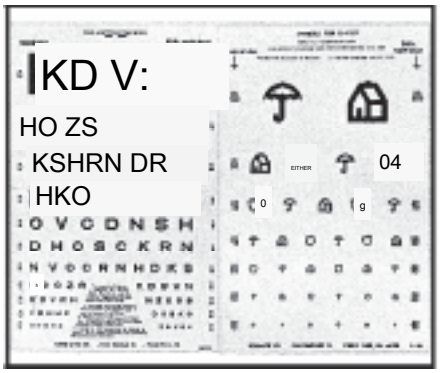


Fig. 15.8 Snellen chart and figures.

A. Exploration of distance vision.

1. Place the person 20 feet away from the Snellen chart, which should already be in place, and ask the person to cover one eye with a cardboard card or cupped palm so that the fingers rest on the forehead and do not compress the eye, while you examine the other eye. It is not appropriate to cover the eye with your fingers, because it could be seen through them and because compressing it can distort vision when it is to be examined. Corrective lenses can be left on, if the subject already uses them, to evaluate if they have the appropriate prescription.

2. It is ordered to read with each eye separately, the letters of different sizes that are in that table, considering maximum vision to be that which corresponds to that line of letters of smaller size that the subject has been able to read without making a mistake.

If the table is not available to perform the non-specialized physical examination, have the headlines of a newspaper or magazine read at a similar distance. Remember to examine both eyes separately.

3. Record visual acuity as a fraction for each eye. Normally the smallest letters on the lines, designated “20" can be read at 20 feet, so visual acuity is recorded as “20/20". The numerator indicates the distance in feet from the subject to the table, which will always be 20; the denominator, the distance at which a normal eye can read the line of letters. This figure is printed next to each line of letters or figures in the table.

If the person wears glasses during the exam, note this in the log:

"Normal distance visual acuity. Vision in both eyes: 20/20 (with or without glasses)".

If the table was not used, record: “Distance visual acuity: roughly normal, reading at about 20 feet from the headlines of a newspaper or magazine.”

Remember to examine both eyes separately.

4. If the individual cannot read any line on the scale, the fingers of the hand are shown to him and he is asked to count them: if he can do so, he is said to have finger-counting vision. If you cannot count your fingers, but see them blurry, you are said to have lump vision. If you cannot even blurry see your fingers, you must be taken to a dark room, and with an appropriate device, project a beam of light onto the pupil and if the subject does not perceive light, it is said that you have amaurosis, anopsia or blindness.

B. Examination of near visual acuity.

1. Ask the person to read the Jaeger chart or small print on a newspaper or telephone directory page, held one foot (30 cm) from their eyes.
2. Record visual acuity for near vision. An unimpaired person is able to read small print at this distance. If the subject has to move the board or paper away to be able to adequately distinguish the letters, he or she is unable to focus on nearby objects due to impaired eye accommodation, which is called presbyopia.

Note: Print 3 of the Jaeger table (J3) corresponds to the phone book print. Print 5 of the Jaeger table (J5) corresponds to the print of the small letters of a newspaper.

Perimetry and campimetry

Perimetry consists of determining the perimeter of the visual field corresponding to each eye, that is, the surface that each eye covers when looking, also called peripheral vision. Campimetry, which consists of specifying the visual field, will be explained in detail in the subject Ophthalmology.

To roughly explore the subject's visual fields, the confrontation examination is performed.

a) Stand in front of the examinee, face to face, looking into each other's eyes in a straight horizontal line at a distance of about 2 feet (60 cm).

b) Ask the person to cover one eye and the observer must close or cover with one hand his own eye that is in front of the one that is not being explored. Both must look at the other's uncovered eye.

c) Fully extend your left arm, if you are exploring the subject's right eye, introduce a moving object or finger into the visual field of the eye being explored, moving your hand along the main axes of the visual field (upper, lower, temporal and nasal) of both, at the same distance from each other, in such a way that when the examinee begins to see it you should also see it at the same time, assuming that your peripheral vision is normal and as long as both look at each other. fixedly, to each other (fig. 15.9).

d) Previously instruct the person to indicate with each movement when he or she sees the finger or the object for the first time and compare the subject's visual field with his or her own.

e) Repeat the procedure with the other eye.

f) Record your findings.

Normal visual fields by confrontation:

- Temporal: extends 90 from the midline.
- Upper: 50°.
-Nasal: 60º.
- Bottom: 70º.

Color vision

You can show the subject some of the simple colors and see if he or she is able to identify them. Specialists have appropriate plates for this exploration, such as Ishihara discs. Examine each eye separately, showing the subject colored objects (red, blue, green and yellow) that may be in the room or showing them pictures with those colors, previously prepared for this examination.

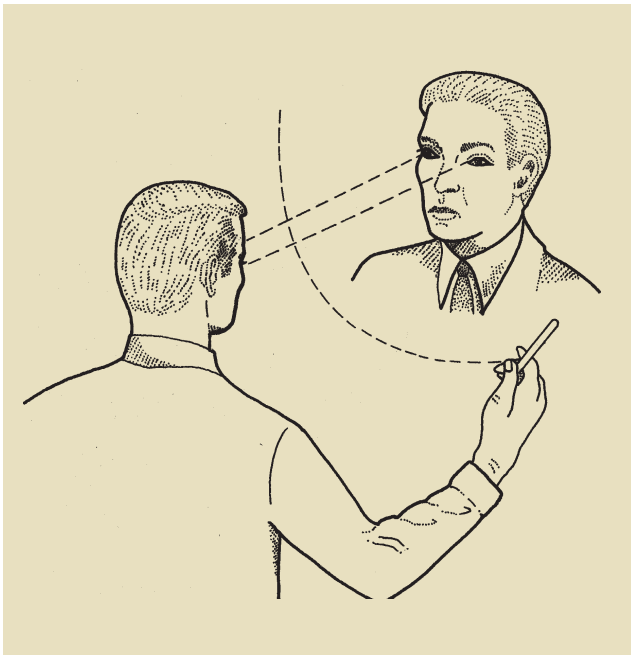
Fundus examination

The examination of the fundus of the eye is performed through ophthalmoscopy, using the instrument called an ophthalmoscopy.

Ophthalmoscope

There are different types and brands, but they all have similar shapes. The handle usually contains the

Fig. 15.9 Examination for confrontation of visual fields.



batteries for the light source, or the cable for the electrical source departs from said handle. They all have a wheel to adjust the diopters of the refracting lenses, which allows a positive or negative number to be seen through a small window. Initially, the focus is set at 0 diopters, which means that the lens neither converges nor diverges the light rays. Depending on both your eyes and the subject's eyes, this device should be adjusted to provide the most accurate focus of the background.

Black numbers are obtained by moving the lens selection wheel clockwise. They have positive values (+1 to +40) and improve viewing if the examiner has difficulty seeing up close. This disk should also be rotated towards positive numbers, to bring the objects in focus closer. If necessary you can use contact lenses or glasses, if the compensation with focus lenses is insufficient.

Red numbers are obtained by rotating counterclockwise, have negative values (-l to -20) and improve visualization if the eXplor has difficulty seeing far away. Some ophthalmoscopes have dials to tune different lens types and apertures. Generally the lens that has the greatest brightness and light emission will be used. The visual aperture can be adjusted by turning the aperture selection dial. To view the different apertures available on your ophthalmoscope, shine the light beam onto a sheet of paper and select the desired lens and aperture.

Usually the largest aperture is selected if the pupils are dilated and the small aperture is chosen if the pupil is constricted. The slit opening can be used to examine the anterior portion of the eye and evaluate the levels of fundal lesions. The gridded aperture can be used to characterize, locate and measure fundal lesions. The red- or green-free filter can be used to evaluate the retina and disc, especially if there is any hemorrhage, which appears black with this filter, while melanin pigments commonly appear gray. The red and green lenses and the slit-shaped or rack-shaped opening are used during special examinations performed by the ophthalmologist.

The internal structures of the eye can be visualized by shining a light source onto the subject's pupil and looking through the viewfinder. The light exits the head through a front glass window.

With the ophthalmoscope, the fundus of the eye appears red-orange, thanks to the translucency through the retina of the blood vessels of the vascular tunic or choroid.

Technique for examination of the fundus of the eye Pupil dilation

In order to see the inside of the eye with the ophthalmoscope, the pupils must be slightly di-canned. Generally this condition is obtained by darkening the room. Short-acting eye drops can be used to dilate the pupil (mydriatics), but it is important to keep a few things in mind before using them. Mydriatics dilate the pupil, because they induce temporary cycloplegia (paralysis of the ciliary muscle) and contraction reflexes to light are lost. Eye accommodation reflexes may also be lost and acute glaucoma may precipitate in susceptible people. The loss of reflexes due to iatrogenic pupillary dilation does not allow them to be examined later, for a variable period of time, which hinders the valuable neurological evaluation of pupil size and its reactivity.

You must get used to trying to see the fundus without using mydriatics from the beginning, to be trained when you need to do so, in the evaluation of a complex neurological situation in which you are involved.
recommended to use them.

A. Explore the luminous red reflection.

1. In as dark a room as possible, instruct the subject to look at a distant point and keep his eyes there, without looking at the light of the ophthalmoscope, while the examination is performed.
2. Hold the ophthalmoscope with your right hand when examining the right eye. Check that the lens is set to zero (0), or adjusted to its diopters, and turn on the equipment light.
3. Stand at arm's length from the person and place the fingers of your left hand on the forehead, so that your left thumb is down on the subject's right eyebrow, holding the upper eyelid. This prevents blinking during the exam, while the thumb serves as a stop when your forehead touches it while bringing the ophthalmoscope closer and does not hurt the subject's eye with the equipment, thus achieving greater stability of the maneuver.
4. From an angle of 15°-30, lateral to the person's line of vision, direct the illumination of the ophthalmoscope toward the pupil of the right eye and look through the viewfinder of the instrument. Notice the red reflection.
   The red reflex is the red-orange coloration of the background, visible through the pupil.
5. As you continue to look through the ophthalmoscope and focus on the red reflection, move toward the person, first obliquely and then frontally, until your forehead touches your thumb, over the person's eyebrow, and you see the red background of the retina.

   Clouding of the lens (cataract) can interfere with visualization of the red reflex. Cataracts appear as white or gray opacities, or they may appear

appear as black spots against the background of the luminous red reflection. Falls vary in size and configuration.

B. Inspect the anterior chamber, lens, and humor or vitreous body.

Inspect the transparency of the anterior chamber and lens. Viewing can be made easier by rotating the lens toward the positive numbers (+15 to +20), which are designed to focus on objects closest to the ophthalmoscope.

The most important abnormalities in transparency that can be found are:

Hyphema (hyphema is correct): appearance of blood in the anterior chamber, which usually results from ocular trauma. Erythrocytes may settle and cause only the lower half of the anterior chamber to appear bloody.

Hypopion: The accumulation of leukocytes in the anterior chamber, causing a cloudy appearance in front of the iris. Secondary to the inflammatory response that accompanies corneal ulceration or iritis.

C. Inspect the optical disc.

1. Now look for a retinal structure such as a glass or the optic disc itself and rotate the wheel to focus the lens with your index finger, until the sharpest focus occurs.
2. If you do not see the optic disc, focus on a glass and follow it in the direction in which it thickens. This will take you to view the disk. Note that the vessels have fewer forks toward the disc.
3. Once the disc is visible, refocus to obtain the best definition.
4. The final setting will vary according to the specific characteristics of your eye structure and that of the subject. If the person is nearsighted, the eyeball will be longer and a negative placement will allow the person to focus further back. Use the positive positions to view through a shorter eyeball, associated with farsightedness.

The optic disc represents the blind spot of the retina.
It is observed towards the medial region of the retinal field.
Normal appearance: round to oval, with very well defined edges; whitish pinkish; approximately 1.5 mm in diameter when magnified 15 times through the ophthalmoscope, from which the blood vessels radiate.
The physiological excavation is slightly depressed and brighter in color than the rest of the disc; The excavation or cup occupies half the diameter of the disc.

D. Inspect the retinal vessels and the rest of the retina.

1. Evaluate the retinal vessels, which are distributed from the disc to the periphery. Four groups of arterioles and veins pass through the disc.
2. Inspect the retinal vessels systematically, moving your line of sight through the major retinal quadrants, using the subject's pupil as the imaginary center of the coordinate axis. Also note the crossing points of the arterioles and veins.
3. Note any retinal lesions while examining each quadrant.

Arterioles: progressively smaller in diameter as they move away from the disc; bright red, with narrow light reflection; 25% smaller than veins; no narrowing or nicks or dents.
Veins: also smaller as they move away from the disc; dark red; no light reflection; occasionally pulsatile.

Arteriovenous relationship: the A-V relationship is the one that exists between the diameter of these vessels. It is 2:3 or 4:5.
Normal appearance of the retina: it is transparent, but shows a diffuse orange-red color coming from the choroidal layer; Pigmentation may be darker in black subjects.
Spots with color alterations, such as white patches, may be abnormal.

E. Inspect the spot.

Towards the lateral region of the disc, an oval field is observed, which constitutes the macula, generally surrounded by several bright spots, with a dark red depression, pit in the center (the fovea). The fovea, the center of the macula, should be approximately two optic disc diameters from the edge of the optic disc. The macula is the point of maximum visual acuity. Examine it last, asking the person to look directly into the light and this places the macula in front of the ophthalmoscope.

Normal appearance: darker than the surrounding background; relatively avascular.

E. Examine the other eye.

To examine the person's left eye, hold the ophthalmoscope in your left hand. Place your right hand on the person's forehead and repeat the examination sequence.

The results of normal fundus examination can be recorded as follows:

Normal transparency media. Intact red reflex. Round disc with defined edges. A-V ratio 2:3. No fundus injuries.

COMMON OCULAR MOTOR NERVES, TROCHLEAR PATHIC AND EXTERNAL OCULAR MOTOR ABDUCENS |I| IV AND VI PAIRS

These cranial nerves are explored together, since they innervate the extrinsic and intrinsic muscles of the eyeball (oculomotor muscles).

ANATOMY AND PHYSIOLOGY

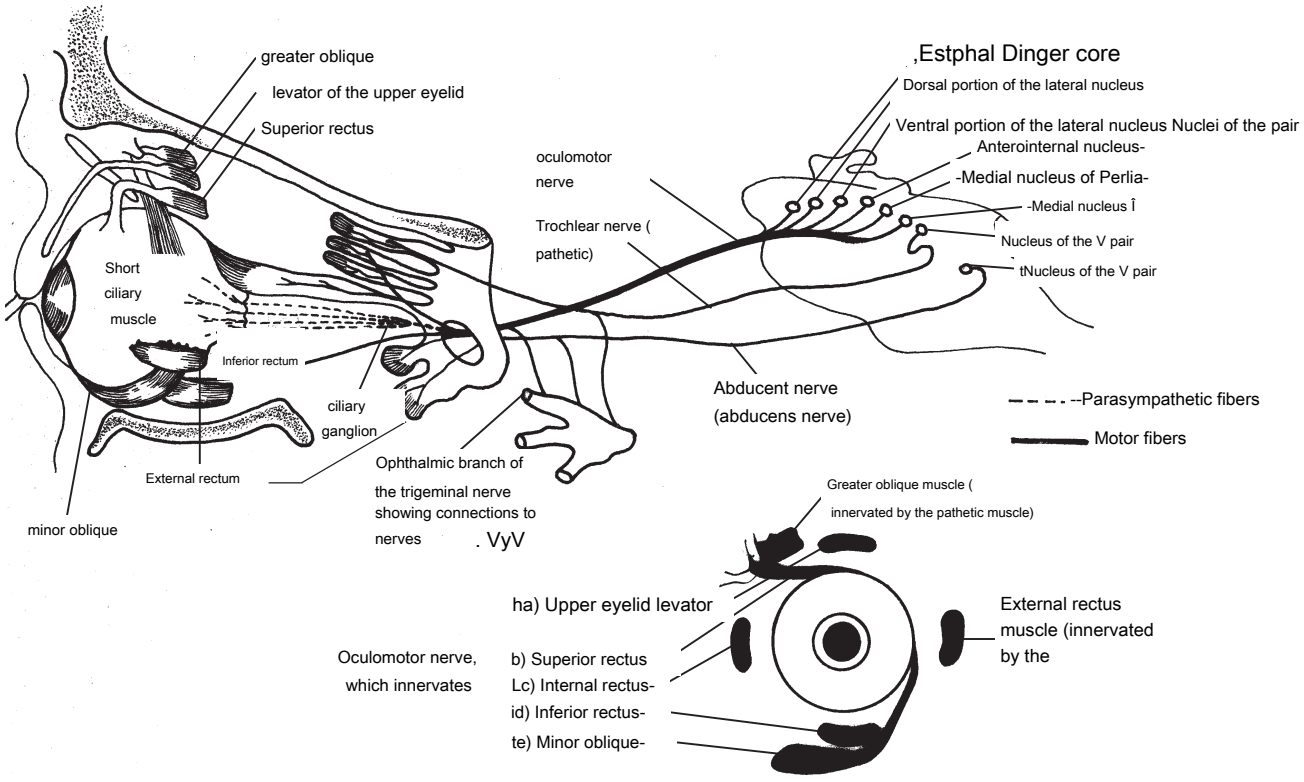
The III pair originates from two efferent or motor nuclear masses that are located in the mesencephalic tegmentum immediately in front of the cerebral or sylvian aqueduct, at the level of the superior quadrigeminal tubercles. The cells that form the nucleus of each of these nerves are of two types:
some somaic and others visceral (the latter are part of the cranial parasympathetic and it is customary to individualize them with the name of the Edinger-Westphal nucleus, to which we have already referred when pointing out the arcs of the photomotor, consensual and accommodation reflexes). The efferent fibers of these nuclei direct forward to emerge on the anterior surface of the midbrain on the inner side of the cerebral peduncle and constitute, one on each side, the oculomotor nerve. Each of these nerves

It directs downwards and forwards, first placing itself in the thickness of the lateral wall of the cavernous sinus, and reaches the corresponding orbit, passing through the jugular foramen on that side.

Immediately upon reaching the orbital cavity, each oculomotor branch divides into two branches: an upper one that innervates the superior rectus and levator palpebrae muscles, and a lower one, destined for the medial rectus, inferior rectus, and minor oblique muscles. Therefore, the oculomotor nerve innervates all muscles of the orbit, except the lateral rectus and superior oblique. Furthermore, parasympathetic preganglionic fibers originating from the Edinger-Westphal nucleus distribute with this pair; They synapse on the corresponding ciliary ganglion and the postganglionic fibers emerging from the latter innervate the constrictor pupillae and the ciliary muscle (Fig. 15.10).

The IVpar originates in the somatic motor nuclei that are also located in the mesencephalic tegmentum, in front of the Sylvian aqueduct, at the level of the inferior colligeminal tubercles. The fibers that emerge from each of these nuclei are directed dorsomedially to, completely intersecting with those of the nucleus on the opposite side, constitute the corresponding trochlear nerve, which has its apparent origin on the dorsal surface of the brain stem.

Fig. 15.10 Oculomotor nerves.


greater oblique
levator of the upper eyelid
Superior rectus
oculomotor nerve
Trochlear nerve (pathetic)
,Estphal Dinger core
Dorsal portion of the lateral nucleus
Ventral portion of the lateral nucleus Nuclei of the pair
Anterointernal nucleus-
-Medial nucleus of Perlia-
-Medial nucleus Î
Nucleus of the V pair
tNucleus of the V pair
Short ciliary muscle
Inferior rectum
ciliary ganglion
External rectum
minor oblique
Ophthalmic branch of the trigeminal nerve showing connections to nerves . VyV
Abducent nerve (abducens nerve)
--Parasympathetic fibers
Motor fibers
Greater oblique muscle (innervated by the pathetic muscle)
ha) Upper eyelid levator
b) Superior rectus
Lc) Internal rectus-
id) Inferior rectus-
te) Minor oblique-
Oculomotor nerve, which innervates
External rectus muscle (innervated by the

next to the midline, immediately below the quadrigeminal plate. This pair, as we see, is the only crossed and dorsally emerging cranial pair. Each trochlear nerve surrounds the brain stem from back to front to be included, together with the cranial base, in the thickness of the lateral wall of the respective cavernous sinus, and reach the orbit through the superior orbital fissure or sphenoid fissure. It innervates the superior oblique muscle on the same side as its peripheral course or, in other words, on the opposite side to that of its nucleus (see Fig. 15.10).

The VI pair has its somatic motor nuclei of origin at the level of the pons or pons, one on each side, immediately below the floor of the IV ventricle;
Its fibers, after crossing the pons in a somewhat ventrolateral direction, emerge at the level of the bulbo-pontuberancial sulcus, above the anterior pyramids of the medulla oblongata. After traveling a short distance next to the base of the skull, they enter the interior of the cavernous sinus on their corresponding side and, crossing the sphenoidal grooves, reach the respective orbit, innervating the external rectus of the eyes (see Fig. 15.10).

In summary, these three cranial nerves innervate all the extrinsic muscles of the eye, the levator palpebrae superioris, the sphincter or constrictor of the pupil and the ciliary muscle.

The nuclei of origin of the three cranial nerves located approximately one below the other, in the midbrain (III and IV pairs) and in the pons (VI pair) are united to exert a coordinated, conjugated action, by means of a formation known as the posterior longitudinal band (medial longitudinal fasciculus) (fig 15.11), and which in addition to uniting the oculomotor nerves to each other, links them with the nuclei of origin of the nerves. vestibular and cochlear (both members of the VIII cranial nerve or auditory nerve) and with the following cranial nerves:

trigeminal (V pair), facial (VII pair), accessory (XI pair) and major hypoglossus (XII pair). In addition, with the motor nuclei of the superior cervical nerves, with those of the posterior commissure (nucleus of Darkshevich) and with the nucleus of the posterior longitudinal band (or interstitial nucleus of Cajal), as well as with some brain centers.

It is an important correlation mechanism, quite intricate, due to the multiple formations that intervene in its constitution, but it allows us to correlate the movements of the head, eyeballs and body. These correlations can be voluntary or in response to various stimuli (visual, auditory, sensory, vestibular, etc.) and give rise to a combined deviation of the head and neck.

the eyes It has great importance in the oculoauditory and oculovestibular reflex functions and in the righting reflexes."

Looking towards different directions in space implies that the movements of the eyeballs are coordinated. For example, looking to the right involves a coordinated action of the right lateral rectus and left medial rectus muscles. These conjugate movements of the eyes are governed by several devices of the central nervous system: the voluntary conjugate movements towards the opposite side, by area eight of the frontal cortex and the conjugate movements that allow following a moving object towards the opposite side, by the occipital lobe. From these brain regions, the fibers are directed to the opposite side of the brain stem and there the posterior longitudinal fasciculus is the association pathway between the nuclei of the oculomotor nerves.

The devices that allow looking up and down and convergence are located in the region of the mesencephalic tegmentum.

EXPLORATION TECHNIQUES

The approach to examination and recording of these three cranial nerves is summarized as follows:

1. Extrinsic eye motility:
   a) Palpebral aperture (II pair).
   b) Eye movements.
2. Intrinsic eye motility (II pair):
   a) Pupils: shape and contour, location, size, symmetry, pupillary hippus.
   b) Photomotor reflex.
   c) Consensual reflection.
   d) Reflection of accommodation and convergence.

Extrinsic eye motility

We will study the extrinsic portion of pair II and pairs IV and VI.

A. Examine the palpebral opening of each eye.

The III nerve innervates the levator palpebrae superioris muscle. Simple inspection of the facies will allow you to realize if the two eyelid openings are of the same width, or if one of them is narrower because the upper eyelid on one side is lowered more than the other (eyelid ptosis) (fig. 15.12). Likewise, if one eye is closed because there is no elevation of the upper eyelid on that side, it will be an index of paralysis of that muscle, due to injury, at least to the third nerve.

Remember that the upper eyelid does not cover the pupil when it opens, but it may cover the upper portion of the iris; The eyelids should open and close completely, without drooping or delay.

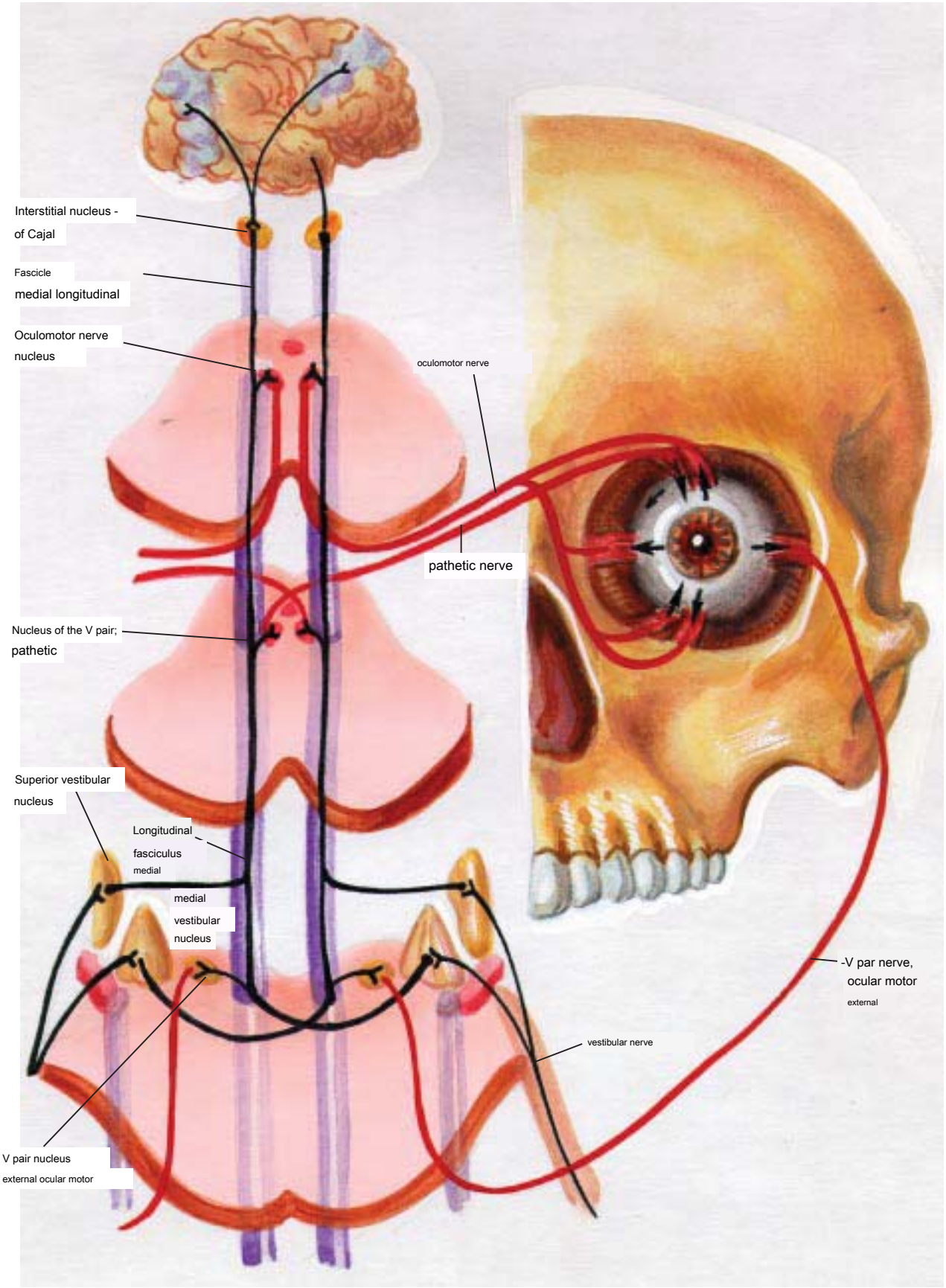


Fig. 15.11 Conjugate eye movements.

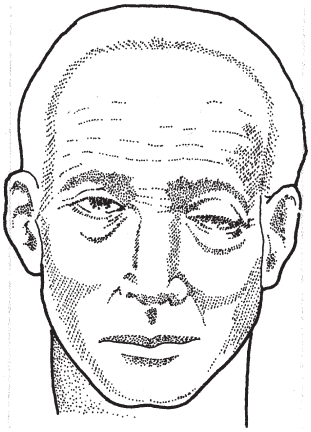


Fig. 15.12 Unilateral eyelid ptosis: combined paralysis of the left II and VI pairs affected by a syphilitic gumma of the lesser wing of the sphenoid.

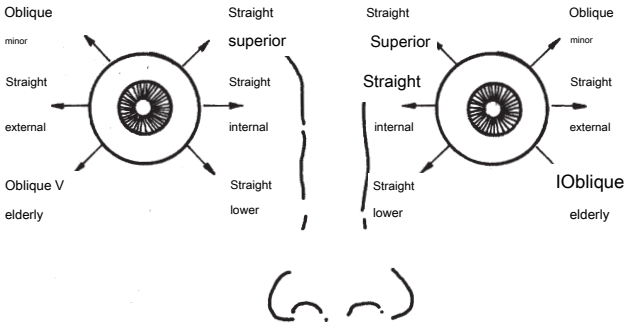
B. Examine eye movements.

1. After observing the palpebral opening, we see if both eyeballs are symmetrical or if, on the contrary, one of them has a deviation upwards, downwards, outside, or inside.

   Normal conjugate gaze: the eyes remain in a central position when at rest.
2. Hold the subject's head with one hand and instruct him to follow a finger, or a pen, that we move in front of his eyes. Move the pen or finger, first in a horizontal direction from right to left and vice versa, to the extreme positions; then, vertically from bottom to top and vice versa. Next, perform the movement in the six directions or cardinal points of the gaze, starting from the center and returning to the central point, which corresponds to the movements that each of the extrinsic muscles impart to the eyeball (fig. 15. 13). We will finish this exploration by moving the finger in a circular direction to give the eyeball a rotary movement.

   Remember that if you move your finger or object too quickly, the subject may have difficulty following you.

Fig. 15.13 Action of the extrinsic muscles of the eye on the movements of the eyeball.



and you cannot adequately evaluate the movements.

cough.

3. When the person looks toward the most distal point in the lateral and vertical fields, note carefully the conjugate movements of the eyeballs and the presence of involuntary, cyclic movements of the eyeball, characterized by a slow initial movement, followed by a sudden jerk in the opposite direction, which is called nystagmus (from nystagmus:
   movement), and whose exploration and analysis we will see when studying par VIII.
   Normal extraocular movements: voluntary movement of the eyes through all positions, without nystagmus. However, slight nystagmus may be observed; It may be non-pathological, when the eyes are in extreme lateral gaze.

4. Examine for strabismus (tape and untape test).

   Ask the person to stare at your pen, held about a foot away, while you cover one of the subject's two eyes. Observe for any movement in the uncovered eye. When removing the cover, observe any movement of the other eye. Repeat the operation covering and uncovering the other eye.

   Normal finding: the gaze remains on the pencil during the cover and uncover maneuver, which indicates good muscle strength and binocular vision.

Intrinsic eye motility

A. Pupils: location, shape and contour, size and similarity
   tria.

In the central part of the iris is the pupil (from the Latin pupilla: girl, apple of the eye), which is a dilatable and contractile opening through which the light rays pass, whose size can be modified by contractile fibers arranged around it, some in a circular shape that constitute the sphincter of the pupil (innervated by the II pair) and whose contraction reduces its size, and others in the form of radii that go from the largest circumference to the minor of the iris, innervated by the sympathetic (ciliospinal center), and whose function is to dilate the pupil.

1. Shape and contour: the pupil is circular in shape and has a regular contour, although sometimes it appears elliptical, and other times, with an irregular contour, which is called dyscoria.

2. Its location is central, although sometimes it may be somewhat eccentric, in relation to the center of the iris.
3. Their size is variable and is related to the intensity of the light to which they are subjected. It expands (increases) in the dark and contracts (decreases) as the light increases. Its average normal diameter is

3 mm and its normal range can be considered between 2 and 4 mm. This varies with age; In the newborn it has its maximum contraction of up to 2 mm: in childhood it acquires its maximum normal dilation: 4 mm and remains in its normal range until middle age, when it progressively decreases, returning to its maximum physiological contraction in old age.

When the pupils are very constricted, less than 2 mm, it is called miosis and when they are very dilated, with diameters of 5 mm or more, it is called mydriasis;

both states are abnormal.

4. The pupils are symmetrical, equal in size. Pupil size inequality is called anisocoria and is generally pathological, although 5% of the population has slight anisocoria, which is considered clinically insignificant.

5. Hippus pupillary. This name is designated to the series of rhythmic contractions that the pupil experiences, either spontaneously or caused by light.
The constant stimulus that light exerts on the pupil and the antagonistic nervous action that is exerted on its constrictor and dilator muscles, mean that the pupil is never completely immobile, but with slight, almost imperceptible movements of dilation and contraction that are called physiological pupillary hippus; When hippus is very evident, it is due to functional or organic alterations that directly or indirectly affect the neurovegetative system.

After observing the characteristics of the pupils and the presence or absence of pupillary hippus (static examination) , the pupillary reactions are evaluated (dynamic examination). This pupillary reaction to stimuli, mainly light, occurs thanks to the innervation of its contractile apparatus that regulates its size: while the parasympathetic fibers of the III nerve contract them, the branches from the cervical sympathetic dilate them. This balance can be broken by irritation of one of the two antagonistic parts and then the effect of this predominates; or due to paralytic injury to one of them and then, the predominant action is that of the healthy part. Example: an irritative lesion of the sympathetic system dilates the pupil, because its action predominates over the constrictive action of the III nerve; The same thing happens when there is a paralytic lesion of the II nerve, in which the pupil also dilates because the antagonistic constrictive action of the III nerve is nullified.

B. Explore the photomoto reflex.

As we already know that intense light contracts the pupil and darkness dilates it, if we direct an intense light beam on it, the pupil contracts; this is called the photomotor pupillary reflex (Fig. 15.14).

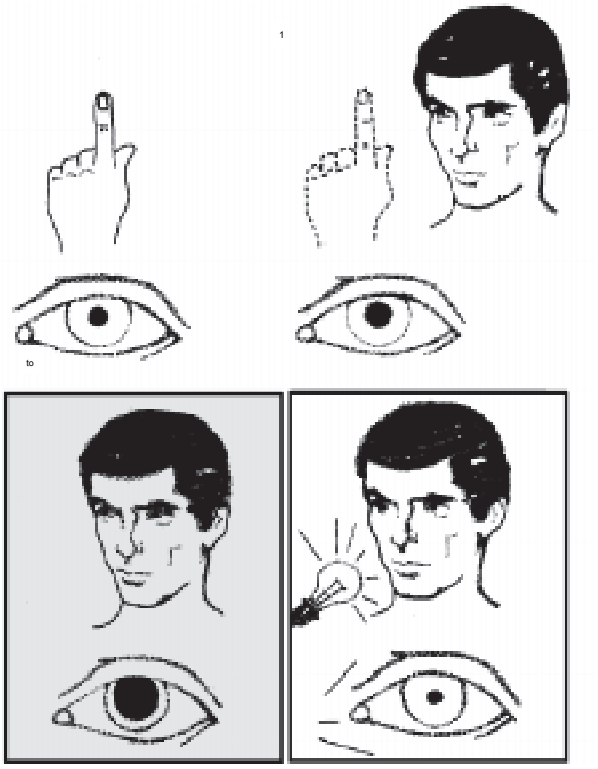


Fig. 15.14 Normal reactions of the pupil: 1, to accommodation: a, in distance vision; b, in close vision: 2, photomotor reflex: a, in the dark (mydriasis): b, in the light (miosis).

1. Darken the room or place the subject with their back to the direct light source.
2. To obtain maximum pupil dilation, ask the person to look at a distant object.
3. Have one eye covered while you shine a beam of light from the side into the pupil of the uncovered eye.
4. Observe if the pupil constricts when the light beam hits.
5. Repeat the test with the other eye.

C. Explore consensual reflection.

When we explore the photomotor reflex, directing the light ray on one eye, we observe that normally the pupil of the other eye also contracts, and that when we remove the light, said pupil dilates; This is called consensual reflex.

1. Shine the light beam laterally on one eye, while observing both pupils.
   Both should contract, even though the light was directed toward only one eye.
   The contraction of the pupil of the eye that does not directly receive light is the consensual response.

D. Explore the accommodation and convergence reflex.

The pupil is also examined by having the subject look at an object at a distance, and then, in front of his or her eyes,

eyes, at a distance of more or less 30 cm, a finger of the examiner is placed, or any object, it is observed that when looking at the distant object, the pupil dilates, and when looking at the finger, the pupil contracts and the optical axes converge. This constitutes the reflection of accommodation and convergence (see Fig. 15.14). The pure accommodation reflex is explored by covering one eye and proceeding in the same way.

Surós prefers the term distance reflection, because the exploration of this reflection consists of looking alternately, from a distant object to a close object, and not as erroneously described in some textbooks and explained by some teachers, of looking at an object held at a certain distance from the subject and bringing it closer until it is 30 cm from the eyes. This would only explore ocular convergence, but not accommodation, because this occurs imperceptibly, as the object gets closer.

On the other hand, if the object is placed far enough away, the observer cannot evaluate the state of the pupils until it gets closer.

TRIG MINO NERVE V PAIR

ANATOMY AND PHYSIOLOGY FIG.

This pair of nerves is the thickest among the cranial nerves. One comes off on each side of the most lateral part of the upper half of the bridge. They are ner-

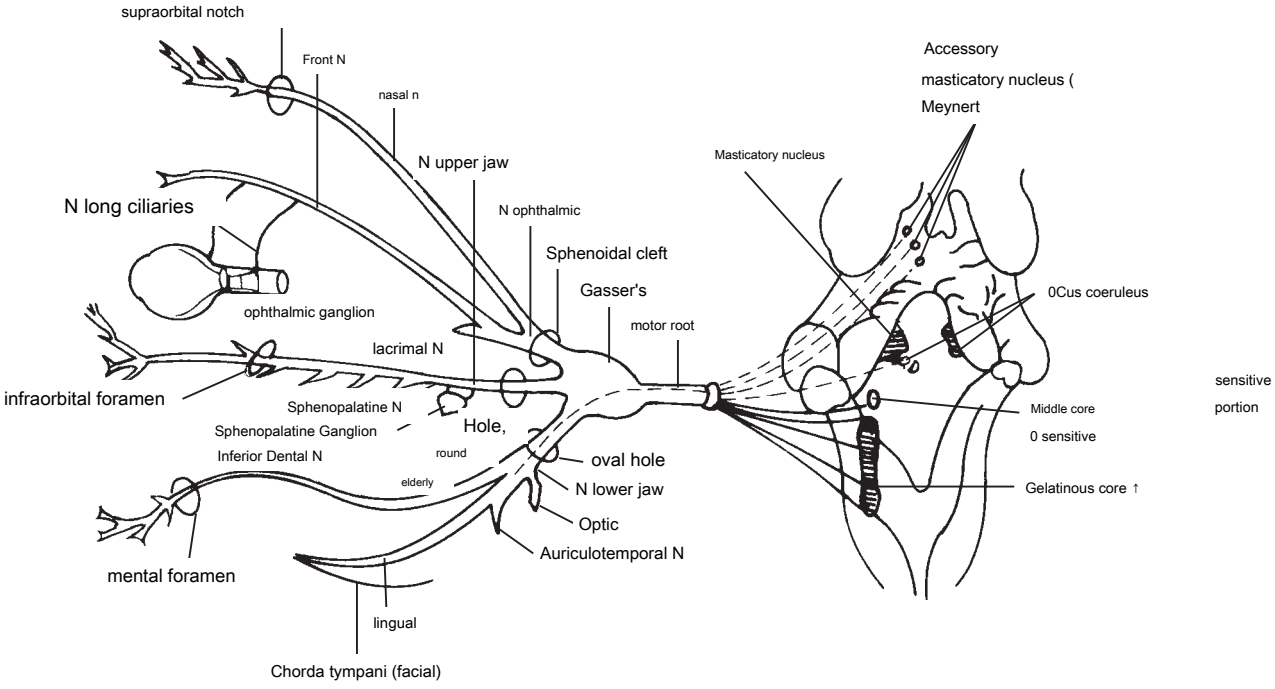
Mixed fibers - so they contain afferent fibers (collected in the so-called sensory root or portio major) and efferent fibers (grouped in the motor root or portio minor).

Each trigeminal, after a short intracranial journey, presents a voluminous afferent ganglion (Gasser's semilunar ganglion), which is resting included in a receptacle formed by the dura mater, on the tip of the temporal petrous bone. The three fundamental trigeminal branches originate from this ganglion: the ophthalmic, the maxillary and the mandibular; These branches leave the cranial cavity through different openings in the floor of the middle cranial fossa: the superior orbital fissure or sphenoidal cleft, the foramen rotundum and the foramen ovale respectively.

Afferent or sensitive portion

It has an extensive innervation territory. The fibers of the exteroceptive afferent (touch, pain, temperature) have their origin in the unipolar cells of the semilunar ganglion; These cells send peripheral processes towards the receptors through the three trigeminal branches and central processes towards the pons. Some (those for pain and temperature) begin a descending path in the brainstem0; Others, the tactile ones, branch into very short ascending branches and much longer descending branches, which together with the painful and thermal branches form the so-called spinal tract of the V nerve, which will extend below to the Lissauer zone of the upper cervical spinal cord segments.

Fig. 15.15 Trigeminal nerve: central nuclei and main branches with their motor fibers (broken line) and sensory fibers (continuous line).



These exteroceptive afferent fibers synapse in the brainstem with a very long nuclear mass rich in cells that extends, occupying a dorsolateral location, approximately from the level of entry of the fibers in the pons to the medulla below (insensibly continuing with the substantia gelatinosa of Rolando of the ipsilateral posterior horn). This nuclear mass is called the main nucleus in its upper part (which is where the tactile and proprioceptive fibers fundamentally synapse), and the spinal nucleus of the V nerve, in its lower part, from the pons to the medulla (it is where the thermal and pain fibers of the spinal tract of the V nerve end).

The axons originating throughout the height of the main and spinal nuclei of the V pair cross the midline at different levels to bend and ascend on the opposite side and form two fibrous bundles: one more anteriorly located, the ventral trigeminal lemniscus; another posterior one, the dorsal trigeminal lemniscus. Both trigeminal lemnisci, independent in their ascending path through the medulla oblongata and pons, fuse in the most superior part of the midbrain to terminate (the fibers that compose them) by synapsing in the posteromedial ventral nucleus of the thalamus on the opposite side to that of their origin. The fibers originating in this thalamic nucleus ascend through the posterior arm of the internal capsule, ending in the postcentral cortex, in the region of the ascending parietal gyrus or postcentral gyrus in its lowest portion.

The trigeminal also contains proprioceptive afferent fibers from the masticatory muscles.
These fibers, in their central course towards the pons, pass through the semilunar ganglion and the sensory root, to reach the bodies of their corresponding unipolar neurons in a nuclear mass that, continuing upwards the main nucleus of the V pair, extends to the dorsolateral region of the upper midbrain: it is the mesocephalic nucleus of the V pair (equivalent to a true afferent ganglion included in the central nervous system).

Although the exact course of the fibers that leave this nucleus to make connections to the brain is unknown, it is believed that they make synapses in the main nucleus of the V nerve, and proprioceptive impulses can reach the thalamus on the opposite side and from here to the postcentral cortex through its fibers.

As we have seen, three nuclear masses correspond to the trigeminal afferent fibers in the brain stem: the mesocephalic nucleus, the main nucleus, and the spinal nucleus. Together they have been classically called the "sensory nucleus of the V nerve" (fig. 15.16).

motor portion

The trigeminal motor nucleus, called the masticatory nucleus by physiologists, emits efferent fibers.

somatic muscles that will innervate, on the corresponding side, the temporalis, masseter, internal and external pterygoid, external peristaphyline, tensor tympani, mylohyoid and anterior belly of the digastric muscles. It is located in the middle third of the bridge, ventral to the main nucleus. The fibers that emerge from it exit the neuraxis through the external side of the ventral face of the pons. In its emergence it makes contact with the sensitive root that we have referred to previously.

The motor root, after a short intracranial journey next to the sensory root, is located below the semilunar ganglion of Gasser (without being included in it), to incorporate its fibers into the mandibular branch, through which they are distributed.

We will roughly indicate the course and distribution of the trigeminal branches:

1. The ophthalnic nerve. Shortly after its exit from the semilunar ganglion, it is located in the cavernous sinus (lateral wall) and, entering the orbit through the superior orbital fissure or sphenoidal fissure, it divides into three branches: nasal, lacrimal and frontal. These three branches receive afferent innervation from: the eyeball, the lacrimal gland, the conjunctiva (except that corresponding to the lower eyelid), the skin of the forehead and the scalp up to the vertex of the skull, as well as the skin of the nose and the upper part of the nasal mucosa.

2. The maxillary nerve. Like the previous one, shortly after its exit from the Gasser's ganglion, it is located on the lateral wall of the cavernous sinus, and when it leaves it it exits the skull through the round foramen; Passing through the pterygopalatine fossa and the infraorbital canal, it emerges on the face below the orbit. This nerve conducts sensitivity to the skin of the upper side of the wing of the nose, adjacent portion of the cheek, lower eyelid and part of the temples, also the lower eyelid mucosa, upper lip mucosa, upper teeth, bony palate, uvula and tonsils, nasopharynx, middle ear and the lower part of the nasal mucosa.

3. The mandibular nerve. After leaving the Gasser's ganglion, it exits the skull through the foramen ovale and shortly after ends in its two branches: the lingual, which after anastomosing with the chorda tympani (see VII pair), is distributed through the mucosa of the anterior two-thirds of the tongue, and the inferior dental nerve, which enters the dental canal through the orifice located on the internal surface of the mandibular ramus. Being a mixed nerve, through its motor fibers it innervates the chewing muscles, already mentioned above, and through its sensory fibers it receives and conducts the sensitivity of the skin on the back.

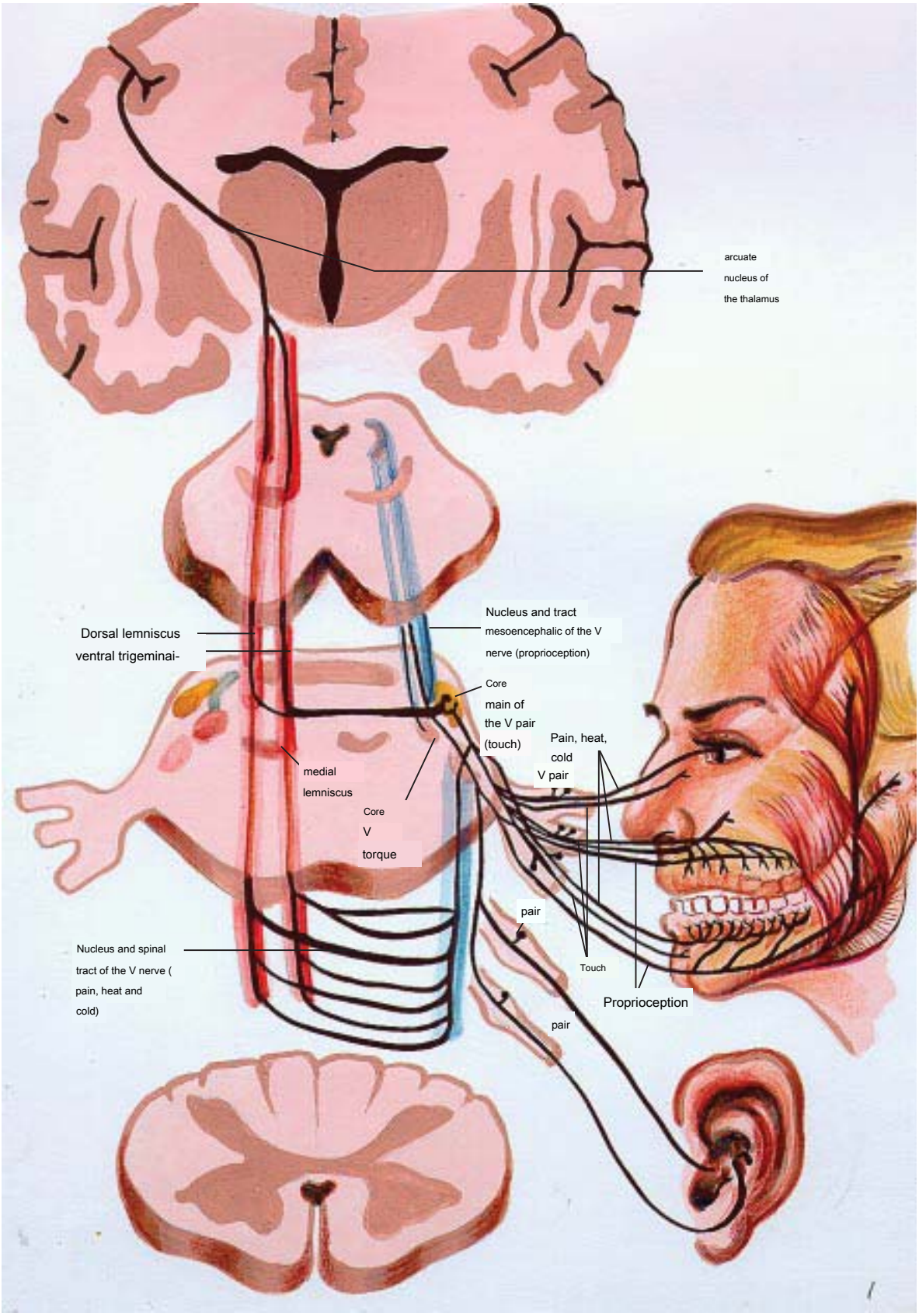


Fig. 15.16 Sensory nuclei and afferent branches of the trigeminal nerve.

from the temples, neighboring part of the pinna, anterior and upper wall of the external auditory canal to the external surface of the eardrum, part of the cheek, the lower lip, the chin, the lower teeth, the inner surface of the cheeks, the floor of the mouth, the anterior two thirds of the tongue (but not with regard to taste, which is dependent on the VII nerve) :

It also contains secretory fibers for the sublingual and submandibular salivary glands, coming from the facial nerve, which it received through its anastomosis with the latter nerve.

Finally, let us point out that, along their journey to the brain, the fibers of the trigeminal sensory pathway establish a series of reflex connections. Among them it is worth mentioning:

1. With the trigeminal mnasticator nucleus on the same side and on the opposite side. This explains the reflex contraction of chewers to percussion of the mandibular, masseteric reflex.
2. With the facial motor nucleus on the same side and on the opposite side. This explains the different reflexes in which the contraction of the orbicularis oculi muscles occurs, due to excitation of: the conea (corneal reflex), the conjunctiva (conjunctival reflex), the eyelashes (ciliary reflex), the skin at the junction of the nose and forehead (nasopalpebral reflex).
3. With the lacrimal nucleus of the facial, which explains the production of tears due to irritation of the conjunctiva (tear reflex).
4. With the salivary nuclei of the facial and glossopharyngeal, which explains the salivary secretion, during chewing or by simple stimulation on the oral walls.
5. With the motor nuclei of the VII and XII pairs, facial and hypoglossal, and even the trigeminal itself, arcs are established for reflexes of importance in chewing.
6. With the nucleus ambiguus of the vagus, with the nucleus of the hypoglossus and with the bulbomedullary centers that intervene in respiratory movements and sneezing (sneezing reflex).
7. With the autonomic or dorsal nucleus of the vagus, which explains the oculocardiac reflex.

EXPLORATION TECHNIQUES

The sensitive portion is explored in a similar way to sensitivity in general; To do this we use cotton wicks, pins, and hot or cold objects (see the exploration of sensitivity in Chapter 14).

Reflexes: corneal (blinking), conjunctival, mandibular and sneezing (they were also discussed in Chapter 14 of this Section).

The motor portion is explored in two ways:

1. Palpate the temporal muscles and then the masseter muscles, while ordering the person to clench their teeth tightly or chew, which allows you to perceive the hardening of the muscle masses due to their contraction.
2. Ask the subject to open his mouth, while opposing it with one hand.

In the case of paralysis of the chewers on one side, we will observe when palpating with the free hand that the masseter on the affected side does not contract or harden, while the masseter on the healthy side will. Furthermore, if the pressure that we oppose to the movement of the mandibular allows it, by asking the subject to open his mouth little by little, we will see that the mandibular deviates towards the paralyzed side, since it is impossible for the muscles on that side to counteract the force of those on the healthy side.

FACIAL NERVE VI| PAIR

ANATOMY AND PHYSIOLOGY FIG.

The facial nerve has four different functions:

1. Somatic motor for the muscles of the face (facial itself).
2. Sensory, responsible for the sense of taste of the anterior two thirds of the tongue (intermediate nerve of Wrisberg).
3. General tenderness for a part of the pinna (Ramsay-Hunt zone).
4. It is part of the cranial parasympathetic (it has secretory and vasodilatory fibers) since it innervates the lacrimal glands, the sublingual and submandibular salivary glands, and the vessels of the mucous membranes of the palate, nasopharynx and nasal passages.

The somatic motor nucleus of the facial is located deep in the pontine cap, in front of the nucleus of the VI nerve. The fibers coming from the nucleus follow a dorsomedial course within the pons and then form a loop around the nucleus of the VI nerve: it is the so-called "knee" of the facial. The fibers emerge from the brain stem through the bulbo-pontine or inferior pontine sulcus.

The facial motor nucleus has two portions: one, upper, and another, lower. The upper portion contains the neurons that innervate the muscles of the upper half of the corresponding hemiface (frontal, superciliary and orbicularis oculi) and the lower portion, the remaining muscles of the face. The superior nucleus receives innervation from both corticonuclear pathways

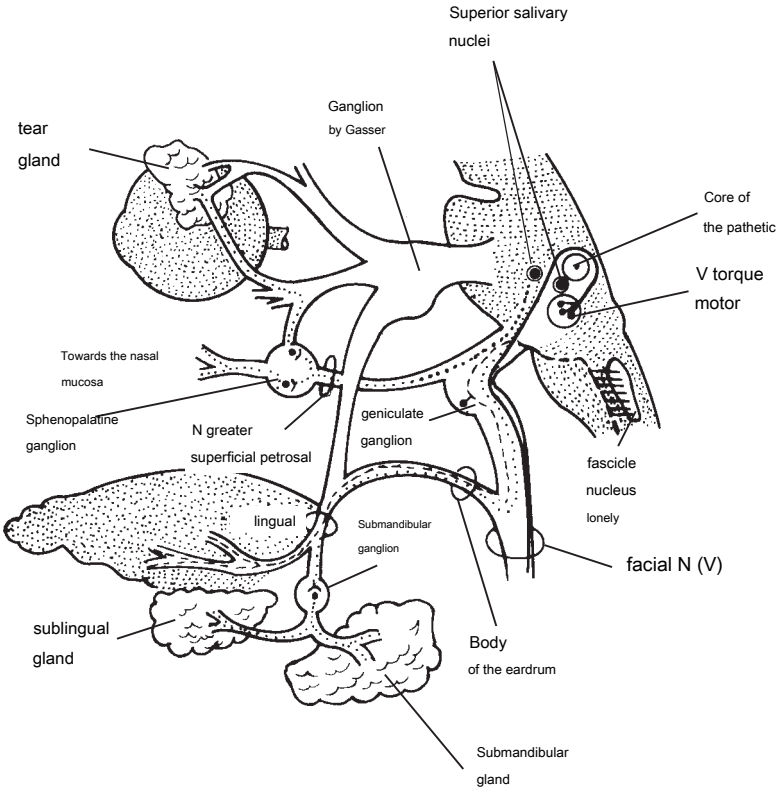


Fig. 15.17 Facial nerve.

(geniculate fasciculus), both from the opposite side and from the same side, while the lower group only receives innervation from the corticonuclear pathway on the opposite side.

The sensory portion of the facial originates in the geniculate ganglion (located in the intrapetrosal tract of the VII nerve), which has unipolar neurons with central and peripheral branches. The central processes form the intermediate nerve of Wrisberg, which enters the stem through the bulbopontine sulcus (next to the motor portion) ending in the upper part of the nucleus solitarius.

The parasympathetic vegetative portion of the VII nerve has nuclei related to the motor portion and the intermediate nerve. Preganglionic fibers originate from the lacrimal nucleus (located very close to the motor nucleus) and run within the facial nerve, through the greater superficial petrosal nerve, then ending in the sphenopalatine ganglion; Postganglionic fibers innervate the lacrimal glands and the designated mucous membranes.

The nucleus that corresponds to the intermediate nerve is the superior salivatory nucleus, anatomically and functionally related to the inferior salivatory nucleus ([X par) and to the nucleus ambigu0, which gives rise to the X par. Preganglionic fibers exit with the Wrisberg intermediate and synapse in the submandibular ganglion. The postganglionic glands end in the submandibular and sublingual salivary glands.

From its emergence in the neuroaxis, the VIth pair goes to the internal auditory canal, then following a path within the petrous portion of the temporal bone and exiting the skull through the stylomastoid foramen; It crosses the parotid gland and finally divides into two branches that innervate all the muscles of the face.

In its intrapetrous portion, the facial innervates the stapes muscle (stapedius) and from it the chorda tympani detaches, an important branch responsible for the taste of the anterior two-thirds of the tongue (it properly includes the peripheral extensions of the ganglion corresponding to Wrisberg's intermediary, already mentioned).

In its extrapetrous portion, the facial gives, among others, the auricular branch, which collects the sensitivity of the pinna (concha, tragus, antitragus, antihelix and a part of the internal auditory canal; known as the Ramsay-Hunt zone.

EXPLORATION TECHNIQUES

motor function

1. Observe from the beginning of the physical examination whether or not there is deviation of a corner of the mouth when speaking or saliva coming out of one side of the mouth.
2. Order the subject to wrinkle his forehead (with this maneuver we explore the upper face), to frown, to close his eyes tightly, to laugh, to show his teeth and to whistle and observe the symmetry.

of the folds, the grooves and the lip corners.

3. Ask the person to project their lips forward while you press against them with your fingers.

4. Also ask him to fill his mouth with air and extend both cheeks. Press them simultaneously with your index fingers and notice if air escapes from one side of your mouth.

5. Explore the strength of eyelid closure by asking the subject to keep his eyes tightly closed, while you try to open them by raising the eyelids with your thumbs.

If the subject is stuporous or in a coma, the Pierre-Marie-Foix maneuver (firm pressure on the posterior part of the angle of the jaws) should be performed, which can reveal lower facial paralysis.

sensory function

We know that the anterior two-thirds of the tongue are sensory innervated by the chorda tympani (facial branch) and the lingual nerve (trigeminal branch). The examination of the sensory function therefore consists of exploring the taste of each hemilenguage, in its anterior two-thirds.

You need to have cotton swabs ready, jars with sugar (sweet taste), common salt (salty), citric acid or lemon juice (acidic) and quinine (bitter), a piece of paper or four cards on which the four primary flavors are written in large letters, and a glass of natural water to rinse your mouth between tastings.

Previously explain to the subject that substances with the four primary flavors will be applied separately to each tongue, that he must keep the tongue out of the oral cavity during the examination of each taste and will indicate with a finger, on the paper or cards, which of the flavors it corresponds to.

First one half of the tongue is examined and then the other.

1. Take a cotton swab, very slightly moistened with one of the substances, so that the flavor does not run, and apply it to the front and middle part of one half of the tongue, reminding the person to keep the tongue out to prevent them from closing the mouth, since the diffusion of the substance can allow the taste in the posterior third.

2. Order him to indicate with a finger which of the flavors it corresponds to.

3. Ask him to rinse his mouth.

4. Repeat steps 1, 2 and 3 for each flavor.

5. Explore the other language in the same way.

6. Record the scan results.

STATOAC STICAL NERVE VII PAIR

The VIII pair or statoacoustic nerve is actually formed by two nerves or two branches: the vestibular nerve, which transmits impulses related to balance and spatial orientation of the body, and the cochlear nerve (sensory nerve) responsible for hearing.

United in a common trunk (a fibrous septum separates them), the two nerves leave the internal auditory canal next to the VII nerve, pass through the cerebellopontine angle and enter the brain stem at the level of the bulbopontine sulcus, immediately behind the VII nerve.

pair.

From their entry into the brain stem their pathways are completely differentiated.

COCHLEAR NERVE

Anatomy and physiology fig

It originates in the spiral ganglion or ganglion of Corti, located in the petrous portion of the temporal bone. Its bipolar neurons send their dendritic processes to the organ of Corti, located in the membranous cochlea of the inner ear. This highly specialized organ captures the vibrations of different sounds through its 30,000 hair cells (approximate number).

The central processes, which form the trunk of the cochlear nerve, synapse in the cochlear nuclei (dorsal and ventral), located in the medulla oblongata in the region where the inferior cerebellar peduncle or restiform body penetrates.

The axons of this second neuron cross and form the trapezoid body. Once crossed, they are located in a fascicle called the lateral lemniscus (or lateral Reil's ribbon) that ascends the brain stem to synapse in the inferior colliculus or inferior colliculus. During its course, the lateral lemniscus synapses with its own nucleus (nucleus of the lateral lemniscus) and with the superior olivary nucleus; It is also related to the reticular substance (see "Sensitivity"). In turn, there are pathways that associate both colliculi or colliculi or colliculi, so hearing has a bilateral reception in the central nervous system.

The colliculi (through the tectobulbar and tectospinal tracts) also connect the auditory pathway with the nuclei of the cranial nerves and the spinal cord, thus acting as a reflex center for the face, body and eyes, in relation to sounds.

Axons originate from the quadrigeminal tubercles that make new synapses with the medial geniculate body, and the fibers originating there pass through the sublenticular portion of the internal capsule and end in the auditory cortex, located in the first gyrus of the

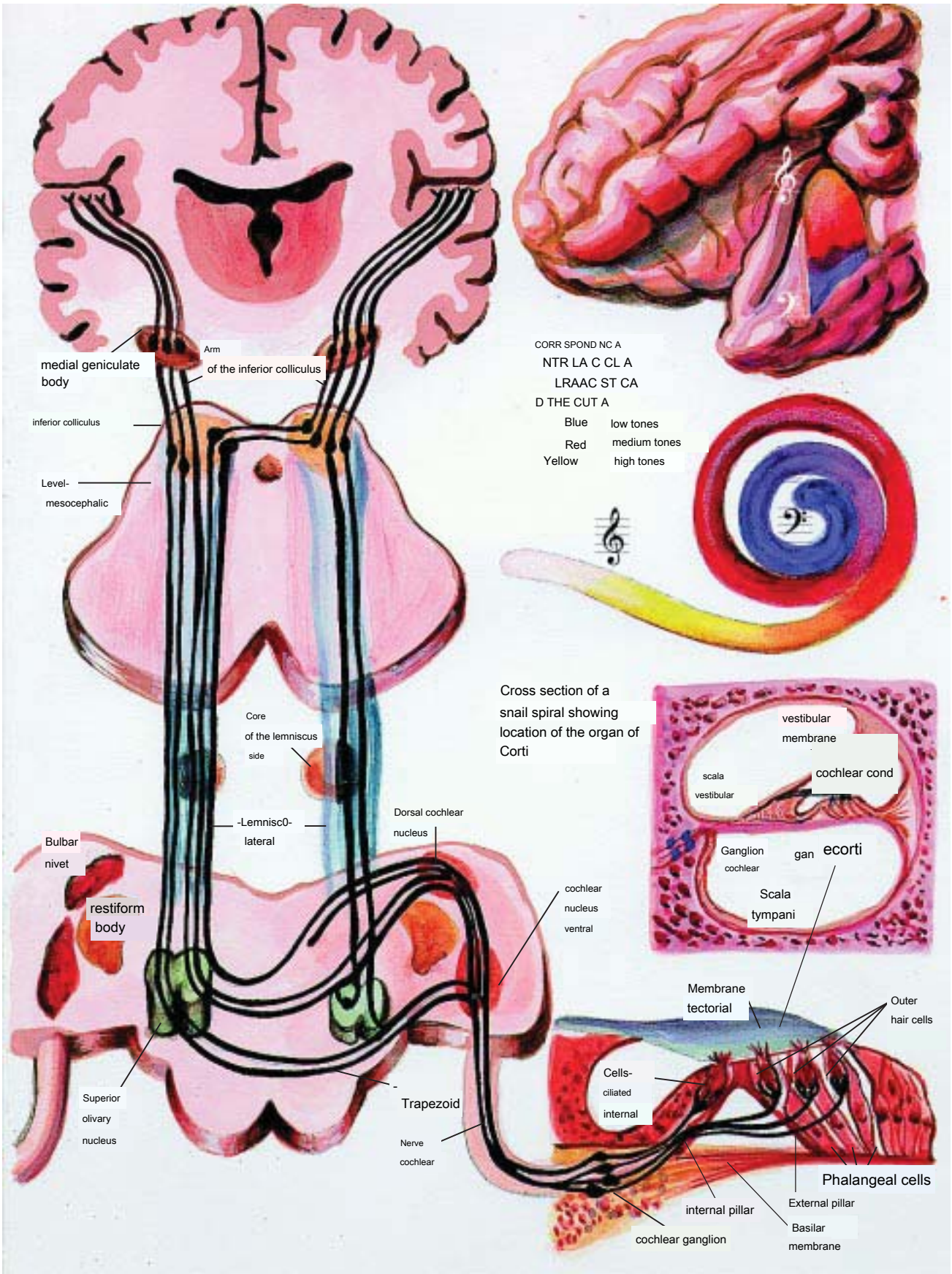


Fig. 15.18 Cochlear nerve (auditory, nerve VIII).

temporal lobe or superior temporal gyrus. Both auditory cortices communicate through the corpus callosum.

From the functional point of view it should be noted that:

1. At the level of the organ of Corti and the first neuron, coding of auditory signals occurs.
2. At the level of the cochlear nuclei, the afferents and their receptor cells are oriented or received, according to the frequency of the sound.
3. At the level of the geniculate ganglion, the tone and intensity of the sound seems to be captured - even before it reaches the cortex.
4. The cerebral cortex integrates sounds in space and analyzes and interprets them.
5. There is a bilateral cortical representation of the acoustic pathway, so unilateral lesions of the temporal lobe do not produce permanent deafness.

Exploration techniques

The examination of the VIII nerve is explained in detail in the subject Otorhinolaryngology. Here we will only give the rudimentary techniques used in taking the usual clinical history.

cochlear portion

Otoscopic examination of the ear. The exploration of the cochlear portion should begin with this exam, which was already explained in the regional exploration, which will allow the subject to observe if there is any obstacle or disease in the external auditory canal, or in the middle ear, by observing the tympanic membrane.

To explore the cochlear portion you need to be equipped with a clock (ticking) and a tuning fork.

Tuning forks. They are used to test for conductive or sensorineural hearing losses. Tuning forks of different sizes generate different sound frequencies. A tuning fork of 512 or 1024 Hz is generally used in examinations, because the human ear can detect frequencies ranging from 300 to 3000 Hz. The frequency number is usually recorded on the instrument.

Activate the tuning fork by grasping it by its stem and striking its end portion against your hand or another surface. Hold the instrument by its stem to avoid dampening vibration.

Explore hearing acuity, especially high-frequency sounds.

In a noise-proof enclosure, occluding one of the external auditory canals, we speak to the person in a low voice at a certain distance, which becomes shorter until the subject hears us.

1. Whispering voice test:

a) Ask the subject to cover one ear with his hand.
Stand slightly behind the person, close to the other ear you want to test.

b) Whisper or whisper a few words and ask the subject to repeat what you have said.

c) Repeat the test in the other ear.

Normally, the subject should be able to recognize the words of the whispered message 2 feet away from the ear being tested.

If he does not hear the whispered voice, a clock is brought closer to his ear, and if he hears the noise of the clock's machinery, it moves further away to determine the distance at which he stops hearing it and then compare it with that of the other ear.

2. Clock ticking test:

a) Stand behind the person. Instruct her to cover the ear that will not be examined.

b) Hold a ticking clock near your uncovered ear. Ask the subject to say "Mr" when he hears the ticking and No when it becomes inaudible. Move the clock until it is 2 feet from the ear.

c) Repeat the test in the other ear.

Note: These two tests do not indicate the subject's ability to perceive low frequency sounds.

If you cannot hear the clock, make a tuning fork vibrate and proceed the same as with the clock. If you do not hear the tuning fork, perform the Weber test.

3. Weber test:

a) Vibrate the tuning fork and place it on the vertex of the skull.

b) Ask the person where they feel the sound and if they hear it in both ears, ask them if it feels louder in one ear than the other.
In normal cases it is heard immediately and equally in both ears, there is no lateralization of the sound (fig. 15. 19).
If it feels more intense or is only heard in one of the ears, the Weber is said to be lateralized to the side where its intensity increases.
When the "transmission system" is affected, bone perception increases and is more intense on the diseased side and when the nerve is affected, perception is more intense, or only occurs, on the healthy side.

c) If the Weber is lateralized, repeat the test occluding first the ear being examined and then the other.

Normally the tuning fork is heard better when the ear has its external auditory canal occluded.
If you cannot hear anything with the ear that has been occluded by the doctor, then we are in the presence of a

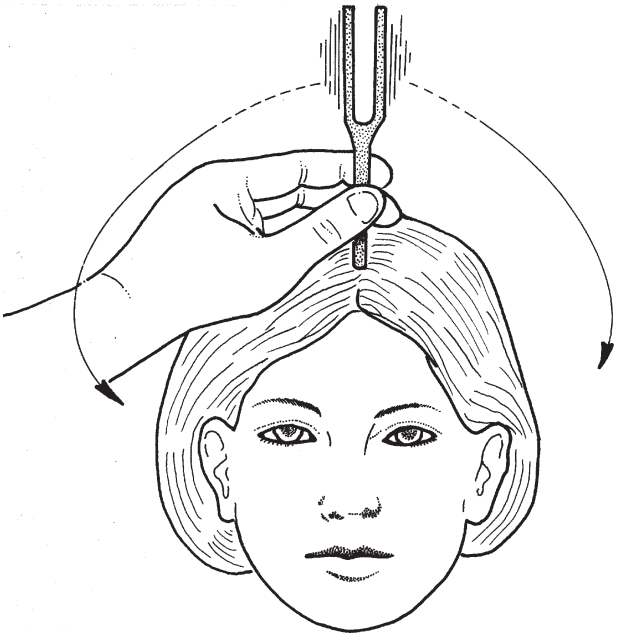


Fig. 15.19 Eber test.

nerve trunk deafness on that side and if the vibrations of the tuning fork are now felt with similar intensity in both ears, it is a bone or transmission deafness, on the non-occluded side.

Tests are then carried out to compare the ability to perceive bone conduction versus air conduction.

4. Rinne test:

a) Vibrate the tuning fork and place it on the mastoid process of the side whose ear we are exploring.

b) Ask the subject to report immediately when the sound (or hum) is no longer perceived.

c) Upon warning, move the tuning fork, which will be vibrating weakly, in front of the external auditory canal.

d) Ask the subject if he or she perceives the vibration again.

Normally the vibration should be heard again when the tuning fork is placed in front of the external auditory canal, since air conduction is greater than bone conduction (ca> co), called positive Rinne.

In middle ear lesions this does not occur, bone conduction predominating over air conduction (co>ca), called negative Rinne.

In lesions of the inner ear and in cases of intense deafness due to nerve causes, the tuning fork is not perceived in either of the two positions in which it is placed.

The practical doctor must come to the conclusion that this test is very basic and only indicative; if he finds any abnormal finding, or even if he suspects it, he should refer the doctor to the doctor.

subject to a specialized doctor, who with the appropriate equipment will be able to perform an audiometric examination and determine exactly the hearing alteration, qualitatively and quantitatively, in each ear.

5. Schwabach test. Measures the duration of perception bone tion:

a) Place the vibrating tuning fork on one of the mastoid processes and measure the time during which the subject perceives the sound.

b) Measure the time on the other mastoid process.

The normal average duration is 18 s; If it lasts less it is said to be "shortened" and if it lasts longer it is said to be "elongated".

c) Additionally, you can repeat the test, holding the tuning fork against your own mastoid and note your bone conduction time, to compare it with those of the examinee, assuming that your hearing is normal.

The purpose of the last three tests is to recognize whether deafness is due to a loss of air conduction (as seen in conditions of the middle or outer ear). In this case, the subject will not hear the clock ticking, the Weber test will be lateralized to the same side as the lesion, the Rinne test will be negative, and the Schwabach test will be longer than normal (more than 18 s) and always on the affected side. On the other hand, if the deafness is due to alteration of bone transmission (as found in lesions of the labyrinth or auditory nerve), it will be seen that the Weber test will be lateralized to the side opposite the lesion, the Rinne test will be positive and the Schwabach test will be shortened.

VESTIBULAR NERVE OR BRANCH

The vestibular branch of the VIII cranial nerve vestibulocochlear (statoacoustic) nerve is responsible for static and kinetic balance and gives us the global position of the head in relation to the various planes of space.

Anatomy and physiology fig

The fibers that make up these branches have their origin in the sensory cells (first neuron) and in the vestibular ganglion (Scarpa), which is located in the internal auditory canal. These first neurons are bipolar cells whose peripheral extensions end in the vestibular receptors located in the membranous labyrinth of the inner ear (utricle, saccule and ampulla of the semicircular canals), whose dendrites are stimulated by the displacement of the endolymph, registering their movements produced by the rotation of the head and captured at the level of the macula of the utricle.

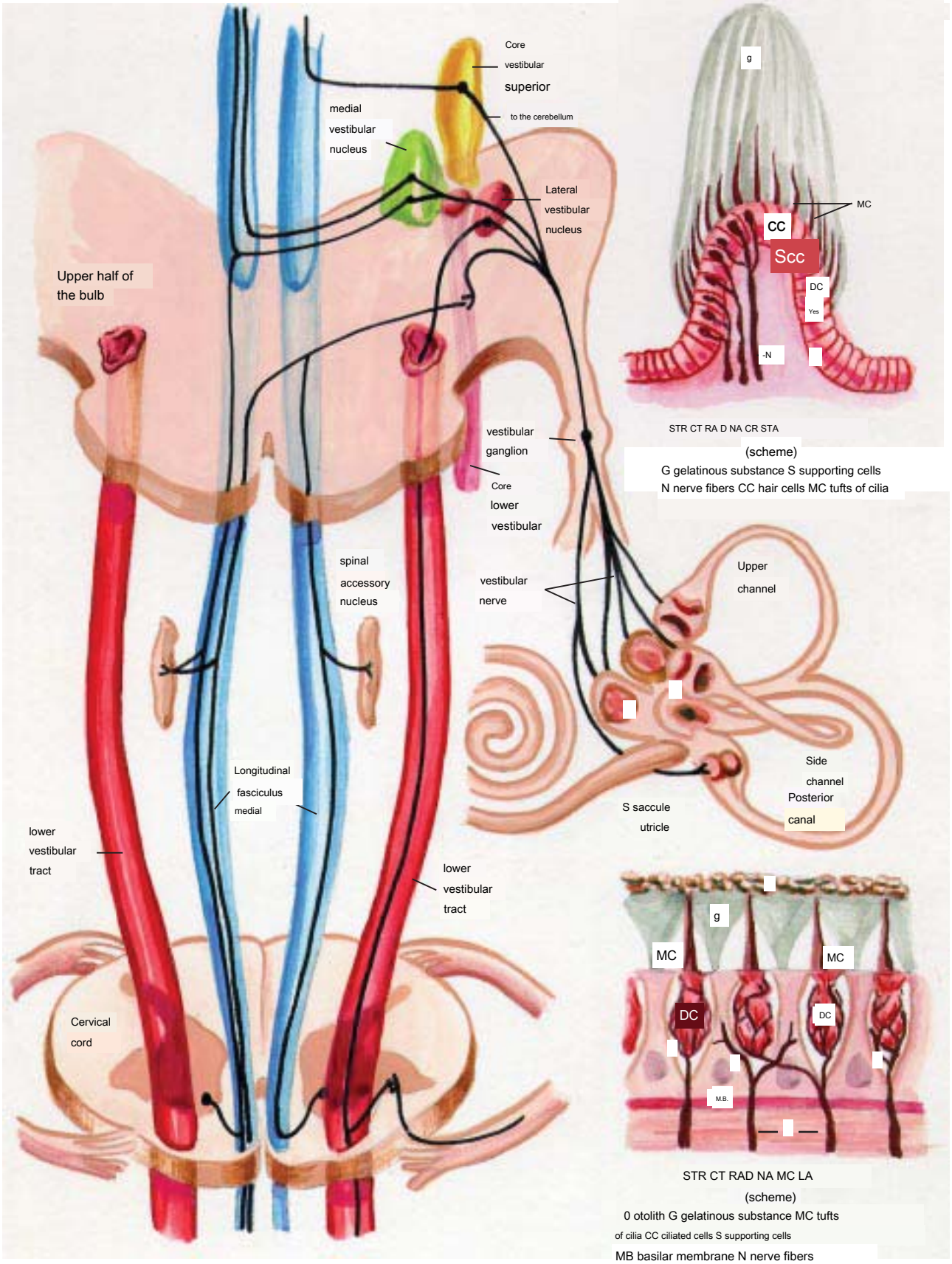


Fig. 15.20 Vestibular nerve (auditory, nerve VIII).

and the saccule and the crests of the semicircular canals, while the central extensions constitute the vestibular nerve that, together with the cochlear branch, reaches the bridge through the inferior pontine sulcus (bulbopontine) heading backwards, towards the floor of the IV ventricle where they divide into ascending and descending fibers.

These ascending fibers terminate in three vestibular nuclei: superior, medial and lateral, while the descending fibers terminate in the inferior nucleus. These nuclei occupy a large part of the floor of the IV ventricle, immediately below the ependyma.

The second neurons of this pathway send their cylindroaxial extensions in different directions and give rise to four important pathways or connections of segmental and suprasegmental interrelation.

1. Vestibulocerebellar connections: from the superior nucleus and reach the cerebellar cortex of the flocculus and nodule.

2. Formation of the medial longitudinal fasciculus (medial longitudinal fasciculus): made up of fibers from all the vestibular nuclei except the lateral nucleus. It extends along the entire length of the brainstem, from the mesencephalic roof to the cervicodorsal segments of the spinal cord.
   The fibers of this bundle end up establishing synapses with the motor nuclei of the cranial nerves that innervate the ocular muscles of the head and neck.

3. Vestibulocortical connections: vestibular impulses reach the cerebral cortex through the thalamic pathway. This path is still debated, since in effect, balance is a reflex act that does not need the influence of consciousness; However, vertigo of vestibular origin are perfectly conscious alterations.

4. Vestibulospinal connections: they originate in the lateral nucleus, descending in the medulla oblongata, posterior to the olive and later, in the composition of the lateral cord of the spinal cord, where they end up synapsing with the intercalated neurons of the anterior horn of the cord.

Features

The vestibular nerves are in contact, on the one hand, with the macules of the utricle and saccule, and on the other, with the ciliated crests of the ampullae of the semicircular ducts. Given the different activities of these two structures, the double static and dynamic function of the vestibular nerve is explained:

1. The static function, on which position reflexes depend, has as its starting point the macula of the utricle and that of the saccule; The macula is ciliated and on them are the otoliths, whose movements are perceived in the movements of the head; The otoliths of the saccule record movements in the frontal plane, and those of the utricle in the sagittal plane. From there the reflexes originate whose destiny is to establish the head in its vertical position.

2. The dynamic function, on which movement reflexes depend, has its origin in the semicircular canals. Such conduits, three in number on each side, are located in three perpendicular planes, one with respect to the other. These ducts contain endolymphatic fluid that, due to its movement, excites the cilia of the ampullary crest. At rest, the endolymphatic fluid is immobile in the semicircular canal.

   During slow movements the displacement of the liquid is damped. On the other hand, when these are intense, the movements are rapid.

Exploration techniques

1. Inspection of the face and eye movements.
   We will observe whether spontaneously or when performing the horizontal or vertical movement towards the extreme positions, a spontaneous movement of the eye appears, characterized by a slow phase and a phase contrary to the previous one, fast, which gives its name to the direction. This is what is called nystagmus. Sometimes it can be explored by fixing the subject's head with one hand and asking him to follow with his eyes a finger of the other hand that is placed in front of his eyes about 30 cm away.
2. Romberg maneuver. Described in the taxi study.
3. Index deviation test, from Bárány:
   a) Stand at arm's length distance from the examinee.
   b) Ask the person to touch the observer's finger with their index finger with their extended arm.
   c) Then ask him to lower his arm, and with his eyes closed, touch the observer's index finger again with his index finger, who, of course, will have maintained the same position he had at the beginning of the test.
   d) Repeat the maneuver with the other arm.
   Normally the subject can do it; In pathological cases it will not do so and will deviate one or both indices in one direction (always the same) during the examination.

4. March. In vestibular disorders the subject will adopt a zigzag gait, deviating to one side or the other.

5. Babinski star. If a person with a vestibular condition is blindfolded and ordered to take ten steps forward and ten steps back several times in succession, it will be seen how he deviates each time.

of the initial line, always in the same direction, as if he were walking following the radii of a star, and being able to finish walking completely with his back to the direction in which he began to walk forward (fig. 15.21).

6. Caloric and rotational tests. They are used to produce changes in the endolymph flow and test the vestibular apparatus. Its technique and interpretation correspond to the Otorhinolaryngology Specialist.

A simpler test is the modified Bárány caloric test, which consists of:

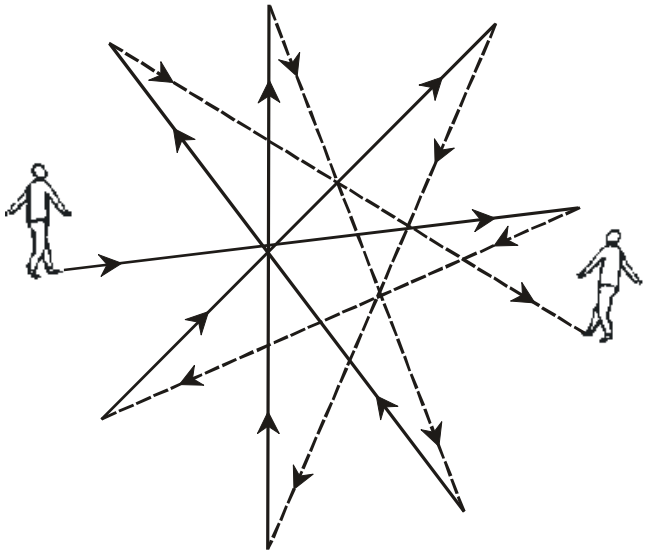
a) With the subject standing and the head tilted 60° backwards, the external auditory canal is irrigated with 100-200 mL of cold water (between 19 and 21°C) or with 56 10 mL of very cold water (0-10°C).

b) The subject is instructed to say when vertigo or nausea begins.

c) Then, the existence of nystagmus is required.

Normal results (irrigating the right ear) are: sensation of vertigo and nausea, horizontal nystagmus with the slow component to the right, fall to the right and deviation of the index to the left. If there is a complete interruption of the vestibular function, there will be no vertigo, nausea, nystagmus, etc., that is, no response. When vestibular irritability exists, the response will be very exaggerated.

Fig. 15.21 Blind gait or star gait, by Babinski.
When walking with eyes closed, the subject alternately takes a few steps forward (solid line) and a few steps back (dashed line), deviating to one side and, as the figure indicates, tracing the profile of a star on the ground along the way.



GLOSSOPHARYNGEAL NERVES PNEUMOG STRICO VAGUUS AND ACCESSORY IX X AND XI PAIRS

General considerations

The student and the general practitioner do not keep in mind certain anatomical details of the three cranial nerves glossopharyngeal, vagus and accessory, which are of the highest interest for the correct interpretation of the semiological manifestations.

The three most important anatomical details are:

Solitary fascicle.

- Ambiguous nucleus.
- Accessory nerve (XI pair, motor).

solitary fascicle

It is a small vertical column located in the substantia reticulata that extends above to near the IV ventricle and below reaches the limits of the sensory junction. The fibers that make it up have their cell body somewhat further inward, where together they constitute the nucleus of the solitary fascicle.

As you can see, there are two formations: one the nucleus of the solitary fasciculus and the other the solitary fasciculus itself. This nucleus of the solitary fasciculus has a sensory function. It gives rise to three nerves; Wrisberg's intermediary, which we already considered when studying the facial nerve (VII nerve), as its sensory branch; the glossopharyngeal (mixed nerve, IX pair) and the pneumogastric (mixed nerve, X pair). The nucleus of the solitary fasciculus apparently derives from the gelatinous substance of the posterior horn of the medulla (through which sensory elements penetrate). It must be remembered that both the nucleus of the solitary fasciculus and the three nerves that derive from it have a sensory function.

Ambiguous nucleus

It is also a small vertical column in the reticular substance of the medulla oblongata that extends above, to the upper end of the bulbar olive and below, until near the sensory junction.

It is nothing more than the representation (at this level of the neuroaxis) of the head of the anterior horns of the spinal cord. Its cells give rise to motor fibers. The nucleus ambiguus is, therefore, a motor nucleus and gives rise to the following nerves: glossopharyngeal (mixed nerve, IX nerve); pneumogastric (mixed nerve, X pair) and accessory (motor nerve, XI pair).

Accessory nerve (XI torque, motor)

This nerve has two sectors of origin: one bulbar, from the bulbar nucleus or most inferior portion of the nucleus ambiguus, which shortly after emerging enters the plexiform ganglion of the vagus (X par) and is distributed with it.

This is what is called the internal root of the accessory or internal pneumoaccessory. Therefore, its study must be done with the vague (X even), since, as we say, they are distributed together. The other sector is the external accessory, with equally motor function, which is distributed by two muscles: the sternocleidomastoid and the trapezius.

GLOSSOPHARYNGEAL NERVE IX PAR FIG.

Anatomy and physiology

The glossopharyngeal nerve is a mixed nerve that, like the VII nerve, contains motor, sensory and vegetative fibers.

The motor fibers start from the nucleus ambiguus, from the highest part of it. From this nucleus, the motor fibers are directed outwards and somewhat forwards to emerge through the most superior portion of the posterior collateral sulcus of the medulla oblongata, between the VIII pair, which is above, and the X pair, which is below. Its emergence is through several fibers that immediately come together in a nerve that heads outward and downward, to the jugular foramen through which it leaves the cranial cavity. At its exit from this hole it has two ganglia: that of Andersch or petrous and Ehrenritter or jugular. It crosses the posterior subparotid space and, attached to the deep surface of the styloglossus muscle, reaches the base of the tongue where it ends.

The only muscles it innervates are the stylopharyngeus and the muscles of the anterior and posterior pillars of the fauces, related to the act of swallowing.

The sensory fibers have their origin (protoneuron) in the cells of the petrosal and jugular ganglia, whose peripheral extensions receive gustatory stimuli from the posterior third of the tongue behind the lingual "y", and sensory stimuli from the mucosa of the pharynx, soft palate, isthmus of the fauces, tonsils, Eustachian tube or auditory tuba and tympanic cavity. The central extensions follow the same path already described for the motor fibers to which they are attached in the nerve, they penetrate the medulla oblongata to end in the middle portion of the nucleus of the solitary tract, located deep and externally in the medulla oblongata, next to the restiform and juxtarrestiform bodies, and in whose upper portion we already saw the gustatory fibers belonging to the intermediary ending.

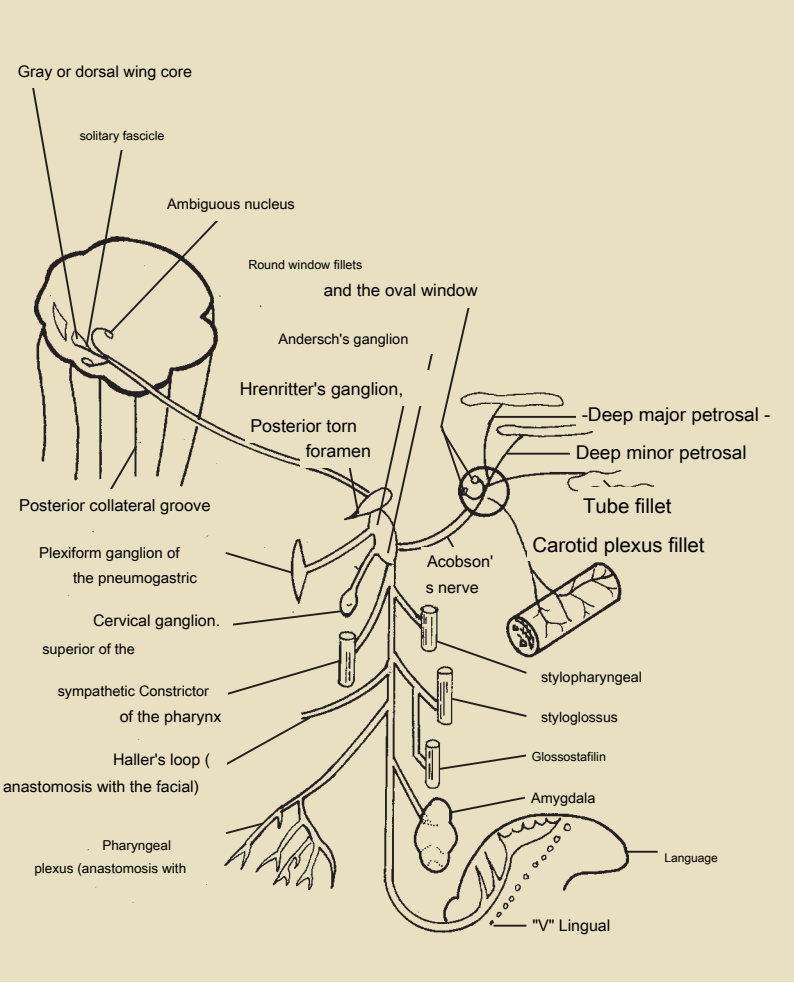


Fig. 15.22 Glossopharyngeal nerve: origin and course.

Wrisberg River (VII pair). This is why it has been proposed to call it the gustatory nucleus. In it, as we saw previously, fibers from the X pair also end up in its lower portion.

Other studies suggest that the fibers that conduct pain sensitivity in the pharynx go through the IX nerve and the tactile fibers go through the X nerve.

The deuteroneurone of this sensory pathway is located in this center. The parasympathetic fibers of the IX pair also have their origin in the medulla oblongata, in a nucleus located deeper, more dorsally and more medially than the previous two, constituted by a set of cells that give rise higher up to the vegetative fibers of the VII pair from the superior salivatory nucleus, which here give rise to preganglionic fibers, which constitute the inferior salivatory nucleus, and which, as we will see when studying the X pair, later on, give rise to the cardiopneumoenteric nucleus of the X pair or vagus whose cells, together, represent nothing more than a long column of visceromotor cells.

From the inferior salivatory nucleus the fibers, following the same course as the nerve, pass through the petrosal and jugular ganglia without stopping there, and through Jacobson's nerve, the tympanic nerve and the lesser petrosal nerve pass to the otic ganglion, where they synapse. Postganglionic fibers originate from this and end in the parotid gland.

Exploration techniques

1. Vernet phenomenon:

a) The subject is asked to open his mouth wide.

b) You are ordered to say "aaaa" while you observe the posterior wall of the pharynx.
Normally, contraction of the posterior wall of the pharynx occurs, which does not occur when the IX nerve is injured.

2. Pharyngeal reflex. Next touch one side of the posterior wall of the pharynx with a wooden depressor or applicator. The normal response is immediate contraction of the posterior wall of the pharynx, with or without nausea.

The IX nerve offers the sensory pathway for this reflex and the motor pathway is offered by the X nerve or vagus; That is why the pharyngeal reflex is shared by both nerves. Normally, the bilateral absence of this reflex is not rare, so its loss is only significant when it is unilateral.

3. Exploration of taste in the posterior third of the tongue. The same technique described above is used for the VII pair, in the anterior two thirds of the tongue. In daily practice, this examination is not performed routinely, due to how uncomfortable it is.

If any alteration is suspected, the specialized doctor performs the examination by applying galvanic current of 0.25 or 0.50 mA (milliampere) to the tongue, which should produce the perception of an acid taste.
If this perception is missing, it indicates ageusia, which is highly suggestive of injury to the XI nerve.

4. Examination of the carotid sinus reflex. Careful pressure that is not very intense or prolonged on the carotid sinus normally produces a decrease in pulse rate, a drop in blood pressure, and if the reflex is very intense, syncope and loss of consciousness of the subject. This reflex must be explored carefully and never without having learned it well.

VAGUS STRIC PNEUMOG NERVE X PAIR FIG.

Anatomy and physiology

The vagus is like the glossopharyngeal, a nerve that contains motor, sensory and vegetative fibers.

The motor fibers arise from the motor cells of the nucleus ambiguus in its middle part, among which they give rise to the IX and XI pairs. From there they head outward and somewhat forward to reach the lateral groove of the medulla oblongata, where they emerge by seven or eight fillets that quickly join together to form a trunk that leaves the skull through the jugular foramen; At this level there is a superior jugular ganglion and a ganglion located immediately below the previous one and called the plexiform ganglion. It then crosses the neck, the thorax, the diaphragm (through the hole for the esophagus) and enters the abdomen, where it ends. The motor fibers of the vagus are distributed throughout the muscles of the soft palate and pharynx.

The sensory fibers of the vagus are of two orders:
somaic and visceral. The somaic cells come from the jugular ganglion cells and through their peripheral branches they receive the sensitivity of the external auditory canal and part of the ear and, through the meningeal recurrent branch of this nerve, the sensitivity of the dura mater of the posterior fossa. Its central extension unites these cells with the spinal tract of the trigeminal nerve and its nucleus.

The visceral ones, born from cells of the aforementioned ganglia, receive sensitivity from the pharynx, larynx, trachea, esophagus, thoracic and abdominal viscera and from a few taste corpuscles that are found near the epiglottis. These fibers are those that carry to the neuraxis the visceral sensations of distension, nausea and impulses concerning the regulation of the depth of respiratory movements and the control of blood pressure. Its central extension unites these cells with the fascicle and solitary nucleus (which we have just studied) and which they contribute to forming, uniting the fibers of the X pair with the groups

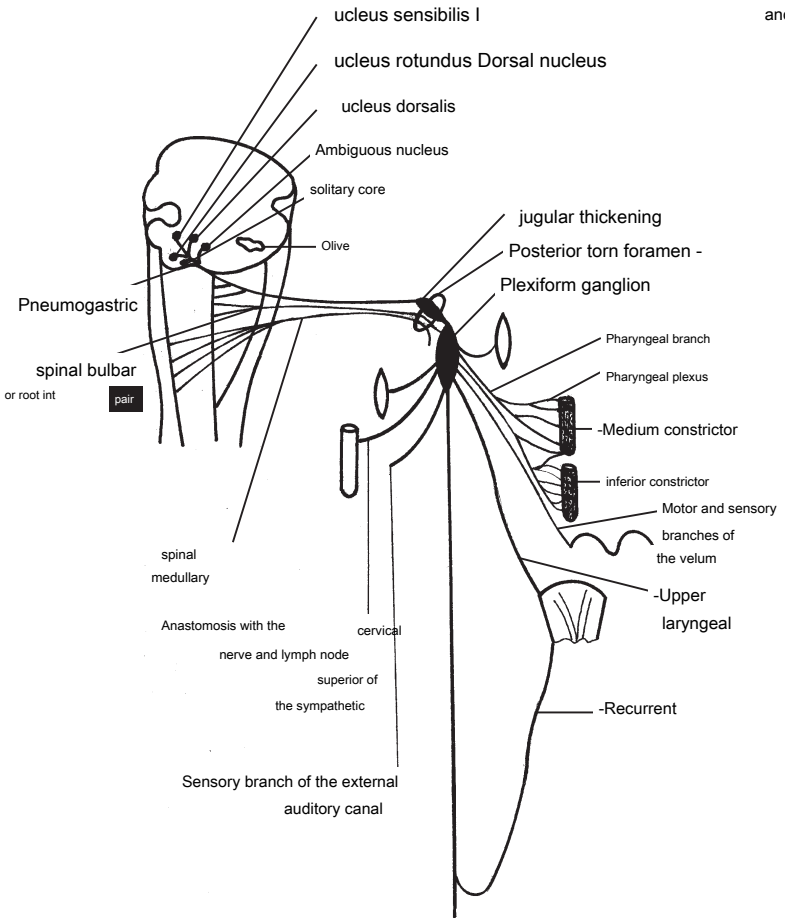


Fig. 15.23 Pneumogastric nerve: origin and journey.

cells located inferior to those that articulate with the fibers of the IX pair.

The vegetative fibers of the vagus are parasympathetic. They come from the dorsal nucleus of the vagus, better called the cardiopneumoenteric nucleus, which we have already seen before, which is the homologue of the body or lateral horn of the medulla; It is a vegetative center and is located at the level of the gray wing of the IV ventricle. The fibers that arise from this nucleus run along the nerve without stopping in any of the two aforementioned ganglia and are distributed through the respiratory, circulatory, digestive and urinary systems. It is important to know that below the emergence of the recurrent nerves, the vagus nerves contain nothing but parasympathetic fibers.

The X pair, like the IX, has a bilateral cortical connection.

Exploration techniques

1. Examination of the soft palate and uvula. Generally, the exploration of the Vernet phenomenon is used for the IX pair, since the technique is the same; What varies is the observation, that instead of focusing attention on the posterior wall of the pharynx, goes the uvula and the two veils, right and left, of the palate.

   When the patient is ordered to say "aaaa" with his mouth open, the velum is normally raised to its full extent and the uvula is kept in the center.

   If there is unilateral paralysis of the vagus, only the velum on the healthy side will contract and, consequently, the uvula will be drawn towards it. The affected side is the same one on which the injury to the vagus nerve occurs.
2. Exploration of the pharyngeal reflex. This is done as described above in par IX.
3. Examination of the carotid sinus reflex. Here what is explored is the vagal component of said reflex; It is done as explained above, in par IX.
4. Exploration of the oculocardiac reflex. With the subject lying supine and with his eyes closed, pressure is placed on the eyeballs with the pads of the thumbs for minutes. The radial pulse has previously been taken and its frequency has been noted.

   After ocular compression, bradycardia should be recorded, the more intense the greater the subject's vagal tone. When exploring this reflection you must have-

Be the same care that we indicated for the carotid sinus.

Its use has been discarded, due to the painful and annoying nature of the maneuver and because it can injure the conea.

5. Examination of the vocal cords using the laryngoscope. Observe if the two cords move, if there is paralysis or paresis of one of the two.
   Examination of laryngeal sensitivity is very difficult clinically.

ACCESSORY NERVE XI PAIR FIG.

Anatomy and physiology

This is a solely motor nerve. The motor fibers that constitute it originate from two different places:
the spinal cord and the medulla oblongata.

From the bulbar nucleus, already known (inferior portion of the nucleus ambiguus), the fibers are directed outwards and somewhat forward, to emerge in the lateral sulcus of the medulla oblongata, immediately below the

The fibers of medulospinal origin constitute the external branch of the accessory. They originate in the cells of the anterior horn of the cord from the first to the fifth or sixth cervical segments. They ascend and constitute the spinal root of the XI pair and enter the skull through the occipital foramen, to join the medullary root and then exit the skull through the jugular foramen. These fibers ultimately innervate the trapezius and sternocleidomastoid muscles on the same side. The nucleus of the XI nerve has bilateral cortical innervation.

Exploration techniques

1. The cervical region and the nape of the neck are inspected, looking for asymmetry or flaccidity of the sternocleidomastoid and trapezius muscles and for atrophy or fasciculations of any of them.
2. These muscles are palpated to check their tone or flaccidity.
3. The subject is ordered to raise both shoulders, with the examiner placing his hands on them and opposing the movement, in order to explore the segmental muscle strength of each trapezius.
4. The subject is ordered to rotate his head, with the examiner opposing the movement, with one hand resting on the subject's chin and observing the muscular force with which the movement is intended to be carried out, and the contraction or not of the sternocleidomastoid muscle on the opposite side.
5. The subject is ordered to flex his head on his chest and resists this movement with a hand on the chin; the head will deviate to the paralyzed side.

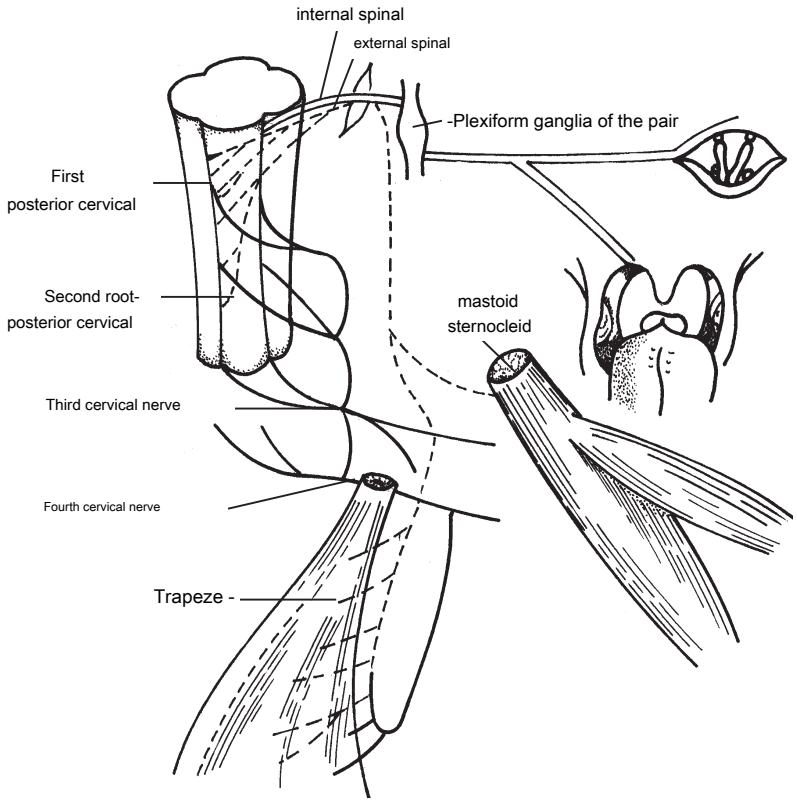


Fig. 15.24 Accessory (spinal) nerve.

IPOGLOS0 NERVE XII PAIR

ANATOMY AND PHYSIOLOGY

It is a motor nerve. Its fibers emanate from two nuclei located in the floor of the IV ventricle: the main nucleus and the accessory nucleus. The main nucleus forms a relief on the floor of the IV ventricle, the internal white wing. These nuclei represent the head of the anterior horns of the spinal cord. From their origin, the fibers travel an intrabulbar path forward and outward to emerge from the neuraxis through the preolivary sulcus of the medulla oblongata, through 10 or 15 vertically oriented threads; Of these, the lowest ones reach the junction of the pyramids, exactly overlapping the anterior root of the first cervical spinal nerve. These fillets meet in two trunks that converge towards the condylar foramen and fuse into a single trunk. Upon exiting the skull, the nerve describes a curve whose concavity faces upward and forward and after passing through the lateropharyngeal space, the carotid and subhyoid regions, it ends on the lateral surface of the tongue, innervating its muscles.

Cortical connections are bilateral.

EXPLORATION TECHNIQUES

1. Trophism and symmetry of the tongue; fasciculations: the person is ordered to open the mouth and the tongue is observed and whether its two halves are equal and symmetrical or if there is atrophy of one of its two halves. The existence or not of fasciculations is also observed.
2. Position of the tongue: the subject is ordered to stick out his tongue and it is observed whether the tip is in the center or deviates to one side. Be careful with false deviations of the tip of the tongue, when there is facial paralysis or when teeth are missing that cause an asymmetry of the orifice at the mouth opening.
3. Segmental muscle strength: Segmental muscle strength of the tongue is tested by ordering the subject to press his tongue against one of the cheeks against which the examiner has placed his fingers or hand on the outside.

GUIDE AND RECORD OF THE EXPLORATION OF THE CRANIAL PAIRS

1. Pair I (olfactory): normal, anosmia, hyposmia, parosmia, cacosmia.
2. Par II (optical): visual acuity (far and near), color vision, pericampimetry, fundus.
3. Par II (common ocular motor) intrinsic portion: pupils: shape and contour, location, size, similarity tria, hippus pupillary; photomotor reflex, consensual reflex, accommodation and convergence reflex.
4. Pair IIII (extrinsic portion), pair IV (trochlear), pair VI (abducens): eyelid indentations: absence of eyelid ptosis. Eye movements: normal.
5. Pair V (trigeminal):

Motor portion	¡Tactile, thermal and painful sensitive portion of the face (examined with general sensitivity) i Inspection and palpation of the chewing muscles: masseter, temporal and pterygoid

6. Pair VII (facial):

Motor portion	Muscles of the face
Sensory portion	Taste in the anterior 2 3 of the tongue

7. Pair VIII (chlochlear vestibule):

Vestibular portion	1Presence or not of horizontal or vertical, spontaneous nystagmus I 0 to the extreme gaze. Romberg Fe Bárány index (explored in the taxi) 1Babinski star: absent
cochlear portion	1 Hearing acuity to whispered voice and ticking clock ¡ Eber maneuver: normal or lateralized Rinne maneuver: positive (normal) or i Sch abach maneuver: normal (18 s), shortened, lengthened

8. Pair I (glossopharyngeal):

motor and pharynx portion	iVernet phenomenon: Imovement of the upper 1 3 of the pharynx, when saying aaaa iPharyngeal reflex Carotid reflex
Sensory portion	Taste in the posterior 1 3 of the tongue

9. Pair (pneumogastric): examination of the soft palate and uvula when saying aaaa. Gag reflex.
Vagal maneuvers (not routinely explored).
Examination of the vocal cords (indirect laryngoscopy by the ENT).
10. Pair I (spinal): strength, tone, symmetry and motility of the sternocleidomastoid and trapezius muscles.
11. Pair I| (hypoglossus): trophism, symmetry, tongue position, fasciculations and muscle strength.

16

PHYSICAL EXAMINATION OF THE DIGESTIVE SYSTEM. BUCOPHARYNGEAL AND ANORECTAL SEGMENTS

Introduction

As will be seen in details in Volume 2 of this book, the digestive system is classically divided into three segments:

1. Superior or oropharyngeoesophageal.
2. Middle or gastroduodenohepatobiliopancreatic.
3. Inferior or enterocoliccorrectal.

The esophagus is not accessible to physical examination; The middle segment, made up of intra-abdominal viscera, is usually not detected in the general examination of the abdomen, unless there is an alteration in the volume of some of its parts and when this happens, it requires the use of special techniques for each organ, which will be studied together with the alterations, in Volume 2; The same occurs with the small intestine and the colon, of the lower digestive segment.

That is why in this chapter we will only study, first, the particular physical examination of the mouth and pharynx, under the name of the oropharyngeal segment, and then, the examination of the rectum and anus, under the name of the anorectal segment.

PARTICULAR PHYSICAL EXAMINATION OF THE OCOPHARYNGEAL SEGMENT0

The human digestive system begins with the oropharyngeal segment, which includes the oral cavity and pharynx.

We will analyze the physical examination of the oropharyngeal segment, after a brief anatomical and physiological summary.

ANATOMOPHYSIOLOGICAL COUNT

Let us remember that when the mouth is closed, that is, with the lips and corners of the lips folded, and we try to unfold them, we find a virtual cavity that is called the vestibule. The vestibule is limited, on the outside, by the lips and cheeks, and on the inside, by the teeth and gums. The limits of the oral cavity itself are: in front and on the sides, the dental arches; behind, the isthmus of the fauces, as a point of passage to the pharynx; above, the palatine vault; and below, the tongue and the floor of the mouth. In this way, the dental arch divides the oral cavity into two parts: the vestibule and the mouth itself.

The oral cavity, including the vestibule and the mouth itself, includes: the lips, with their faces and labial glands; the cheeks or buccal mucosa; the gingiva or gum, with its own characters; the teeth;
the palatine vault, with its hard and soft portions, and in this, the uvula; the tongue, with the taste corpuscles:
and the floor of the mouth. We must also consider, as annexed elements, the salivary glands and, as part of the pharynx, the palatine tonsils.

Except for the teeth, the visible structures of the oral cavity are covered by a mucous membrane.

Oral mucosa. The oral mucosa keeps the mouth hydrated, collaborates with digestion and serves as a mechanical and chemical barrier to trauma and infectious organisms. This begins on the inside of the lips (labial mucosa) and covers the inside of the mouth, especially the cheeks (jugal mucosa); It is composed of three layers: the epithelium, the lamina propria and the submucosa (fig. 16.1).

The epithelium, the superficial layer of the mucosa, is separated from the lamina propria by a basement membrane. Both the buccal mucosa and the rest of the oral, pharyngeal and esophageal mucosa are covered by a stratified squamous epithelium similar to the skin, which gives them resistance to changes in temperature and to foods or foreign bodies of greater or lesser hardness. The underlying epithelial cells undergo progressive differentiation as they migrate from the basement membrane to the outermost layers. Rapid cell growth and differentiation occurs in the epithelium and the epithelial surface is renewed between 7 and 14 days. The final differentiation state of the cells varies in different parts of the oral cavity. For example, epithelial cells on the tongue evolve to form taste buds, which contain taste corpuscles, while the epithelium of the lips and cheeks become cells that secrete saliva.

The lamina propria is made up primarily of fibrous connective tissue that lies beneath the epithelium.
Blood vessels and nerve endings extend through this sheet.

The deepest layer of the oral mucosa, the submucosa, is the interface between the mucous membranes and underlying structures, such as muscles and bones. The submucosa of the roof of the oral cavity or palate is thin, which allows it to withstand mechanical aggression and is thicker on the lips, cheeks and tongue, which gives them a soft and spongy appearance.

Blood flows through the submucosa and lamina propria, and is responsible for the pink color of the oral mucosa. It may become redder when inflamed and bluish (cyanotic) with hypoxemia.

Gingiva and teeth. The gingiva or gum is the highly vascularized and innervated oral mucosa that surrounds the necks of the teeth, adhering them to the maxilla and mandible.

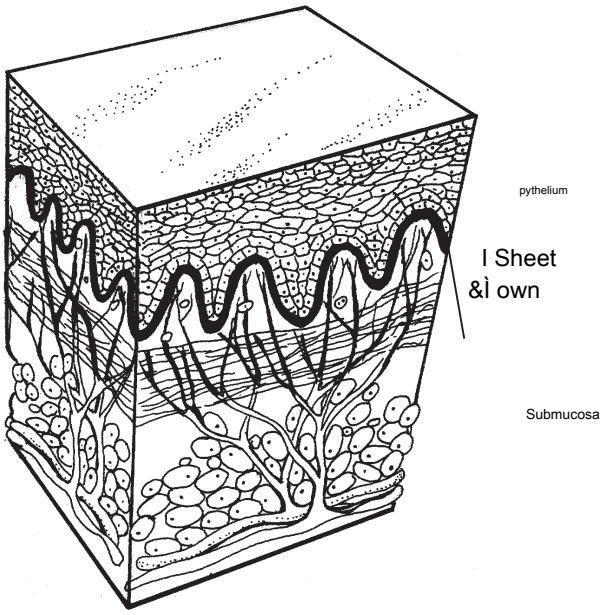
The gingiva is thick and dense, because it lacks submucosal tissue.

An adult must have 32 permanent teeth, distributed symmetrically, 16 in the upper or maxillary arch and 16 in the lower or mandibular arch.

Each of the four hemiarchs consists of eight symmetrical permanent teeth: two incisors, one canine, two premolars and three molars; named by their form or function, from front to back: medial incisor, lateral incisor, canine or cusp, two premolars or bicuspids, first molar or 6-year molar, second molar or 12-year molar and third molar wisdom tooth (fig. 16.2) .

It is important to know that currently the dentigram for recording teeth consists of four quadrants and teeth are recorded by numbers, instead of names. The quadrants are numbered, starting with the upper right hemiarch and in the same direction as the movements of the clock hands, quadrant two corresponds to the upper left hemiarch, three to the lower left hemiarch and quadrant four to the lower right hemiarch. The teeth are numbered in each quadrant, from 1 to 8 and from front to back; so that the medial incisor will be 1 and the third molar will be 8. For example, if the canine of the upper right hemiarch is missing and a cavity is detected in the first molar of the lower left hemiarch, it is reported as: missing 13 and cavity in 36. The first digit is the quadrant and the second is the number of the tooth.

Fig. 16.1 Layers of the oral mucosa.



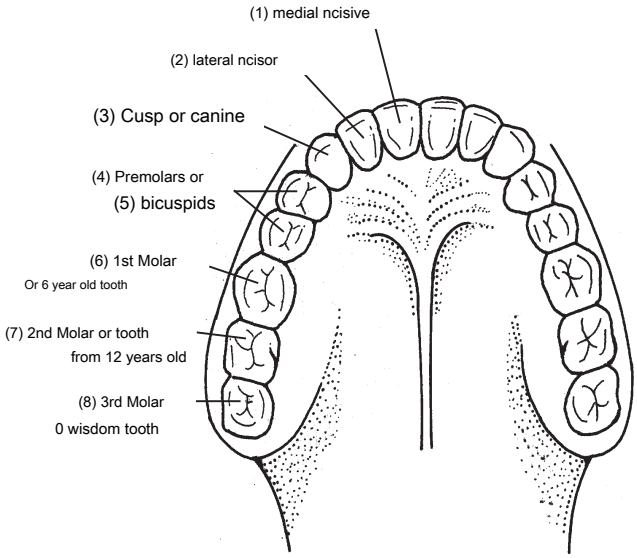


Fig. 16.2 Denture and hard palate.

In children, for non-permanent or "baby" teeth, which are only 20 (5 in each hemiarch), to differentiate the dentigram from that of an adult, the quadrants are numbered from 5 to 8, following the same order;
In this way, quadrant number l of the adult is 5 in the child and 8 of this corresponds to 4 of the adult. For example, the absence of the lateral incisor of the upper left hemiarch would be recorded as 62.

All of the above facilitates the recording of data and the terms are unified internationally, both for medical and stomatological language.

The teeth facilitate digestion, cutting, crushing, grinding and mixing food, in coordination with the chewing muscles of the jaw, which are innervated by the V cranial nerve (trigeminal) and the VII cranial nerve (facial).

The exposed portion of the tooth, or crown, is covered by enamel, the hardest substance in the body. Even so, the enamel can be invaded by a transparent and discolored mass of bacteria and debris, called dental plaque, which when it falls off leaves a hole commonly called a cavity.

Tongue and taste bulbs. The tongue is a movable musculomembranous apparatus, which occupies most of the oral cavity; It is the main organ of taste and contributes to chewing, swallowing and articulating sounds.

It is composed of intrinsic muscle fibers (lingual muscles) and extrinsic, coming from the hyoglossus, geniusglossus, styloglossus and palatoglossus muscles, which together form a mass divided into two halves by a fibrous septum (lingual septum).

The gingiva is covered by a mucous membrane that has a fold or frenulum on the lower surface that fixes it to the floor of the mouth; This membrane has four types of papillae: filiform or conical, at the tip and edges; fungiform, on the anterior part of the back;
lenticular or goblet-shaped that form the lingual "V", and hemispherical, behind it (fig. 16.3).

Gustatory bulbs are found mainly in the calciform and fungiform papillae (Fig. 16.4). Each taste bulb is made up of 20 taste epithelial cells. The microvilli of taste cells extend along the surface of the tongue; They are projections that collect the sensory stimuli of taste. Destruction of the microvilli can cause degeneration of the taste bulbs and a subsequent decrease or absence of the sense of taste.

Ebner's glands, at the end of the tongue, supply serous secretions that distribute substances over the taste corpuscles.

Taste epithelial cells undergo rapid synthesis and differentiation and are highly vulnerable to chemotherapeutic agents, which rapidly destroy dividing cells.

Although all taste bulbs have some ability to distinguish taste sensations: sweet, sour, salty, and bitter, these four sensations are associated with different areas of the tongue. For example, the gustatory bulbs specialized in detecting sweetness are located in the anterior tongue, while the bulbs that detect bitterness are located in the posterior tongue. The bulbs for the four tastes are located on the lateral edges and the salty taste is detected on the entire surface of the tongue.

Taste perception may decrease with age.
The sense of taste is influenced by smell, and taste may be diminished or absent when smell is altered.

The major hypoglossus is the motor nerve of the tongue.
Taste sensations are conducted to the cerebral cortex through the sensory portions of the VII nerve (facial) and the IX nerve (glossopharyngeal). The sensory function in the anterior portion (located in front of the lingual "V") depends on the lingual nerve, a branch of the lower jaw that, through the chorda tympani, receives the fibers of Wrisberg's intermediate. The base of the tongue (located behind the lingual "V"), as well as the soft palate, the pillars of the soft palate and the posterior wall of the pharynx, receive their innervation from the glossophagus. ryngeal.

The tongue coating results from the deposit, between the papillae, of exfoliated epithelial cells kneaded with saliva, food residues, mucus, bacteria and fungi.

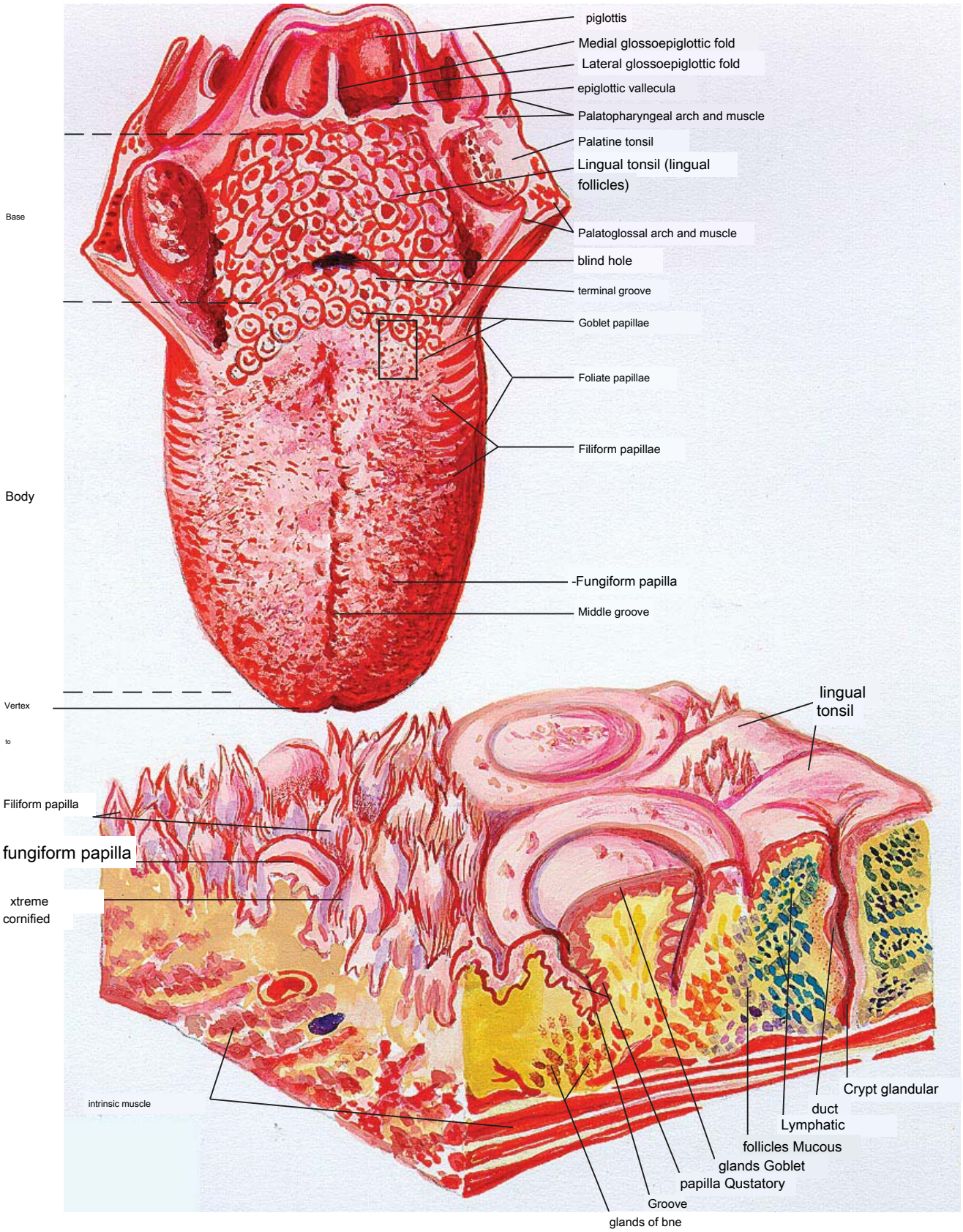


Fig. 16.3 Anatomy of the tongue: a, its dorsal surface to the epiglottis; b, Schematic stereogram of the area boxed in (a).

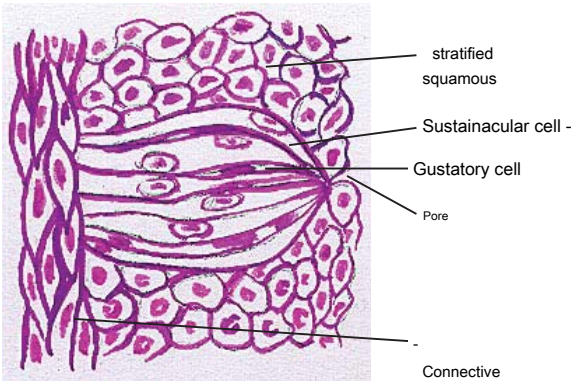
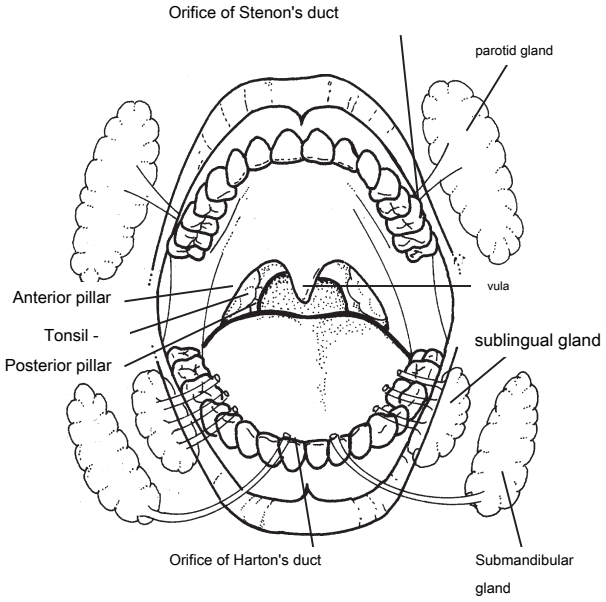


Fig. 16.4 Section of a gustatory bulb.

A small amount of coating is normal on the back of the back, which varies according to the diet. Without lingual papillae there is, therefore, no coating.

Salivary glands. Consisting of a number of glands, within or near the oral cavity, that secrete saliva, including the parotid glands, located under each ear, the submandibular and sublingual glands, on the floor of the mouth, and the buccal glands, in the epithelium of the lips and cheeks (fig. 16.5). The opening coming from the parotid gland opposes the second molar of the upper dental arch, and is marked by a small papilla, called Stenon's duct. The openings of the submandibular glands, or Wharton's ducts, are located under the tongue, at the base of the frenulum, the structure that joins the base of the tongue to the floor of the mouth (fig. 16.6). Each sublingual gland has about 20 ducts, which open on the surface of the gland, at the base of the mouth.

Fig. 16.5 Salivary glands and their ducts.



Under normal circumstances, 1,500 mL of saliva is produced in 24 hours. The production rate varies from 0.2 mL/min, when the glands are at rest, to 4 mL/min during maximum activity.

Saliva production is regulated by the salivary nuclei, located in the brain stem, which are stimulated by certain tastes, smells and tactile sensations, and even by thoughts about food. Excessive salivation can be associated with nausea, because the salivary nuclei are very close to the center of the vomit.

Saliva cleans the oral mucosa, gives it a moist and shiny appearance, and provides protection against bacterial infections in several ways. First, the pH of saliva (6.0-7.0) keeps the environment balanced for the normal bacterial flora of the mouth. If this balance is broken, uncontrolled growth of one or more organisms may occur, possibly leading to infections. Second, substances in saliva, such as lytic enzymes and secretory immunoglobulin A, also inhibit bacterial growth.

In addition to protection, saliva has an important digestive function: it contains enzymes that initiate the breakdown of starch.

Palate or palatine vault. In addition to forming the roof of the mouth, the palate separates the mouth from the oropharynx and is divided into the hard palate, the anterior two-thirds, and the soft palate, the posterior third. The hard palate assumes a convex configuration and is covered by a thick, pale mucous membrane. The soft palate, covered with a thin red mucous membrane, extends to the uvula, a free-hanging structure at the entrance to the pharynx. The soft palate is flexible and changes shape when the mouth is relaxed; It moves upward during swallowing, closing the nasopharynx and pharynx and preventing food from being aspirated into the respiratory tract.

Oropharynx. The oropharynx sits between the soft palate and the epiglottis. The palatine tonsils, small masses of lymphoid tissue, are located behind and below the uvula, on each side of the oropharynx, under the pharyngopalatine arch. The epithelial tissue covering the tonsils invaginates to form superficial depressions or crypts. Tonsils are normally the same color as the surrounding mucous membranes.

EXPLORATION OF THE OCOPHARYNGEAL

SEGMENT General

The equipment necessary for optimal exploration of the oropharyngeal segment includes: adequate artificial light source, at least one pen-type flashlight; tongue depressors; examination gloves or finger cots; gauze size 10 x 10 cm; and a dental mirror, if available.

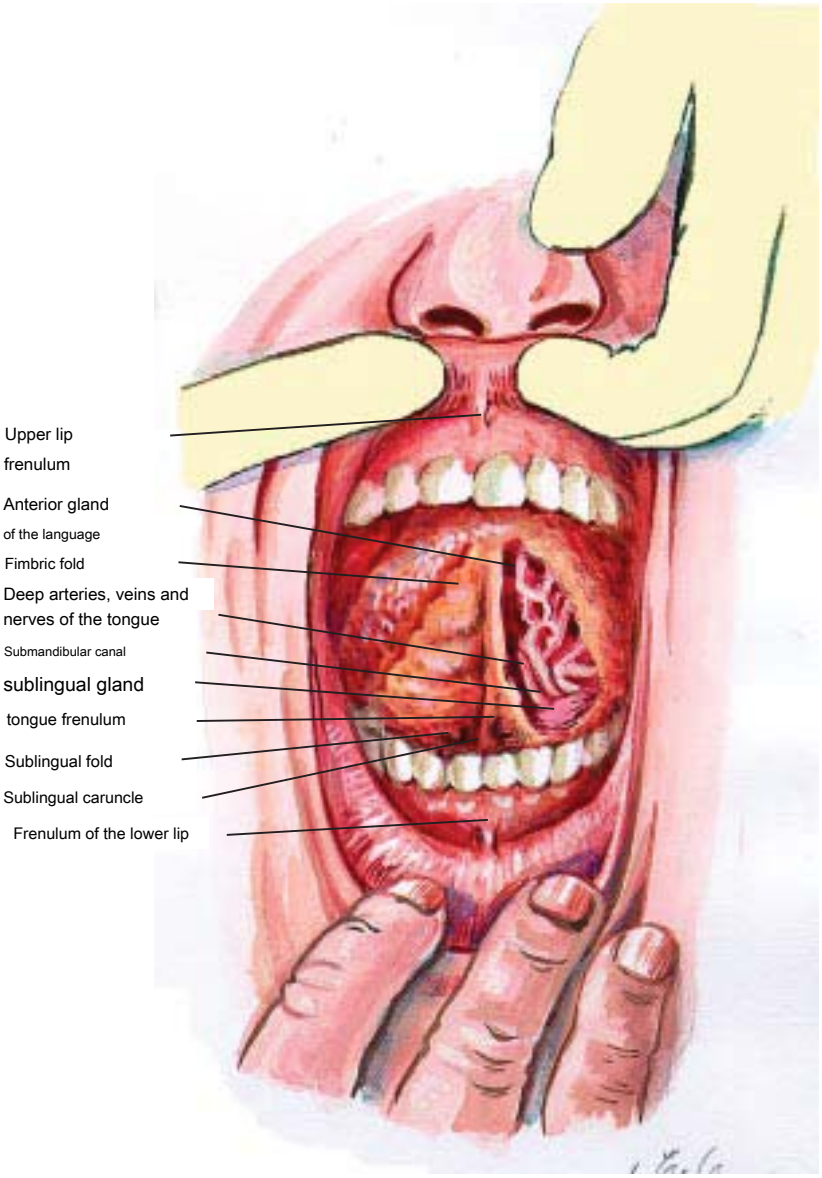


Fig. 16.6 Ventral surface of the tongue and floor of the mouth.

The basic examination technique used is fundamentally inspection, and when there are lesions, it is complemented by digital palpation or digital touch, with a gloved hand, which allows a better appreciation of the consistency, shape, volume, sensitivity, etc., of those.

To carry out the inspection, take the following into account:

Before the exam, explain to the person what you are going to do, to gain their trust and cooperation; Ask them if they have noticed any previous abnormalities and ask them to remove them if they have any dentures. Wash your hands well, before and after the examination, if possible, in front of the subject to be examined.

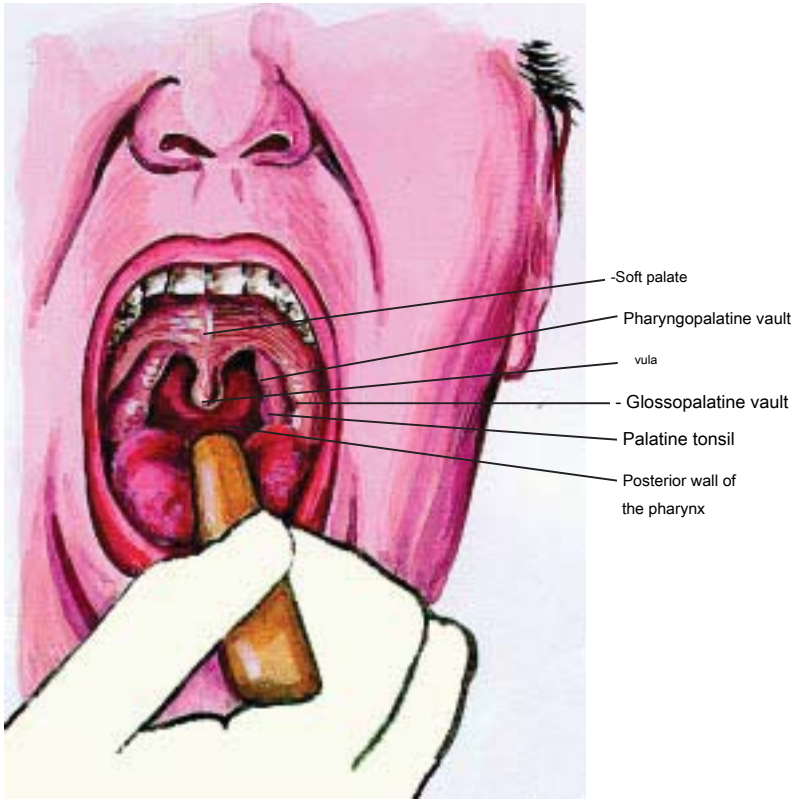
Illuminate the oral cavity with natural light, placing the person in front of a window, so that they receive the light from the front; you stand with your back to it

ma; Then, invite the subject to open the mouth widely, and insert the tongue depressor, as necessary.
To facilitate inspection, move the tongue with gauze or the depressor and use a dental mirror, if available.

The depressors in common use are made of metal and must be sterilized from one subject to another, or they can be made of wood; in this case, they are discarded after use. By depressing the tongue, the pharynx, the isthmus of the fauces and the base of the tongue can be better observed (Fig. 16.7).

In children, you must use the depressor, and sometimes also the mouth opener, or insert one or two fingers at the level of the last molar, to immobilize the jaw. When the child does not cooperate, the examination must be carried out with the help of an assistant who, sitting with the child between his legs, puts one arm around the child's chest, and with the hand of the other arm holds the child's head back,

Fig. 16.7 Tongue depression for examination.



and so you can examine it freely. When you are going to depress the tongue with the depressor, to see the posterior part of the oropharynx, rest it against the teeth, to fix it and maintain its stability, and depress only the anterior half, because if the depressor touches the posterior third of the tongue, it usually causes the gag reflex.

You can also use artificial lighting, which consists of the use of front mirrors, which reflect the light from a wall bulb on the region to be examined, or also by using the illuminating mirror itself or a pocket flashlight.

The examination method must be very orderly: first, examine the external structures of the oral cavity, first with the mouth closed to observe possible alterations of the lips, corners, etc., and then, with the mouth open, or in movement, to explore dental occlusion, the temporomandibular joint and the masticatory muscles. Also examine the salivary glands, at the beginning or end of the oral examination.

Then, observe the mucosa of the lips, the cheeks or buccal mucosa, the soft palate or soft palate and at the level of the gums, to specify the color and lesions that may occur at those levels.

Then examine the teeth, noting the number, shape, interdental spaces, possible presence of lesions, enamel color, etc.

Next, examine the tongue in its different parts, with it inside and outside the mouth; face first upper dorsal, detailing the base region and forcing the tongue out through the tip, if necessary;
Then, inspect the edges and the underside, and ask the subject to make the corresponding movements of the tongue: to one side and the other, as well as to touch the roof of the mouth with the tip of the tongue, to see its ventral side.

Next, examine the hard palate and soft palate, including the uvula or uvula, and the movements of both.

The examination of the mouth includes the examination of the oral pharynx or second portion of the pharynx (oropharynx) , the pillars, the tonsils, as well as the Waldeyero ring, a circle of lymphatic formations, which protects against infections at that level.

The exploration of the sense of taste and the cranial nerves related to the oropharyngeal segment were already studied in Chapter 15 of this Section.

Exploration techniques

external structures of the oral cavity

1. With your mouth closed, inspect and palpate the lips and look at the corners of your lips.

   Explore the symmetry, shape, color, volume, and existence of lip and corner lesions.
2. Ask the person to show their teeth and open and close their mouth to assess malocclusion and the range of voluntary movement. Also ask him to

perform lateral movements with the jaw. As the mouth opens and closes, palpate the temporomandibular joint with the index and middle fingers of both hands, looking for increased volume and deviation of the jaw. Take the opportunity to palpate the chewing muscles, which are part of the exploration of the motor portion of the V cranial nerve, already studied.

The upper and lower teeth must be aligned and occlude the mouth properly with the bite.

The full range of motion of the temporomandibular joints is that in which the mouth can open and close completely and the jaw can move from side to side.

3. Inspect the salivary glands to discover possible alterations:

a) Parotid: in the parotid regions, below the ears and behind the vertical branch of the maxilla.

b) Submandibular: below and inside the horizontal branch of the lower jaw itself.

c) Sublingual: in the mental region, under the chin.

4. Palpate the salivary glands externally, to collect information about the pain, consistency, mobility of the glands and lumpomas (term used by the Argentine school) or tumors at that level.

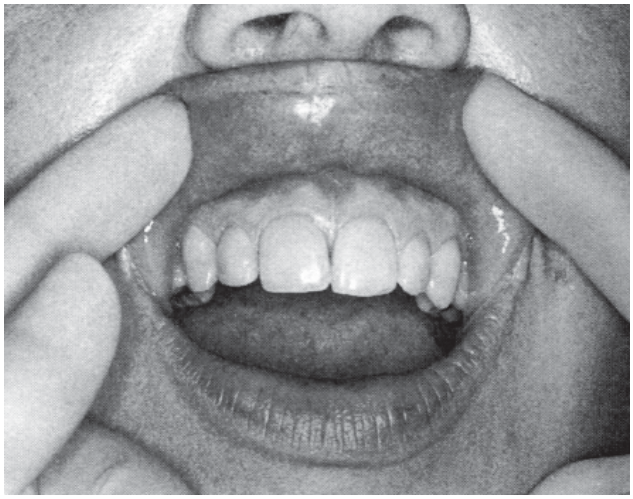


Fig. 16.8 Examination of the labial mucosa and gingiva.

xmen of the oral cavity

Labial, gingival and cheek (jugal) mucosa

1. Examine the labial mucosa, on the inner side of the lips, and the gingival mucosa, on the anterior surface of the gums, moving the lips with gloved fingers or gauze (Fig. 16.8).
2. Explore the inner cheek or buccal nucosa, using a depressor or a gloved or finger-coated finger to move the cheek laterally and expose the surface (Fig. 16.9).

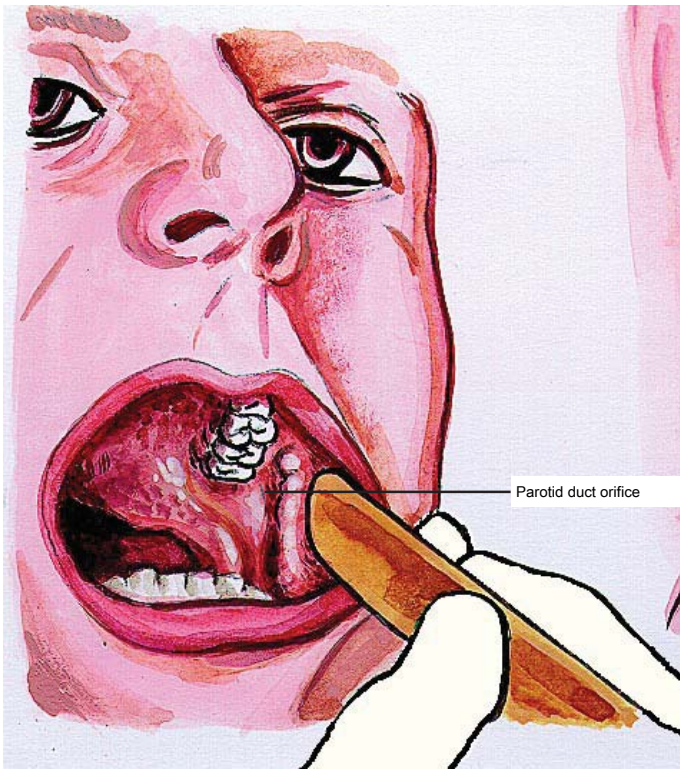


Fig. 16.9 Examination of the buccal mucosa and Stenon's duct.

3. Look for the Stenon's duct (of the parotid) on each side of the cheek, which is located just in front of or at the level of the second molar of the upper jaw.

The color of the lips and cheeks varies with race, bluish red in blacks. The rest of the oral mucosa is pink in all breeds. Although rare, in blacks there may be hyperpigmentation in the oral mucosa; This is smooth, moist, free of lesions and exudates.

Teeth

Logically, although the thorough examination of the teeth belongs to the field of the stomatologist, the examiner can, through simple observation, identify dental diseases and refer them to this professional.
On the other hand, there are systemic diseases that cause changes in the teeth; others have their cause in dental diseases; Furthermore, the absence of teeth can contribute to facial asymmetry, affect speech articulation or tongue movements, which can be misinterpreted as neurological alterations.

According to the normal number of teeth according to the age of the subject, look for numerical anomalies, eruption (early or late), size, shape and structure, enamel color, implantation or location, interdental spaces, fillings and possible presence of lesions, such as plaques and cavities.

Language

In ancient times, inestimable value was attached to the examination of the language. The English especially said that "the tongue was the mirror of the stomach", however this later fell into certain discredit; However, the observation of language has largely recovered its importance, stripped of the empiricism of that time.

and supported by certain scientific bases. From the embryological point of view, it is clear that it cannot be considered the mirror of the stomach or the rest of the digestive system, since its epithelium is of ectodermal origin, while the rest of the digestive system, with the exception of the anus, is of endodermal origin.

1. Examine the dorsal surface of the tongue, first in place and then ask the person to protrude the tongue (Fig. 16.10).
2. Observe its size and volume, its shape, color, appearance, moisture, lesions, fascicular movements, position with protrusion and symmetry of the tongues.
   The normal tongue is of size and volume, in accordance with the space reserved for it in the oral cavity. When the tongue is well papillated, the taste buds give its dorsal part a slightly rough, uniform texture. Its color must be similar to that of the mucosa that surrounds it, although it may be more whitish, in the presence of a tongue coating, and it must be moist, without fascicular movements, in a central position with the protrusion and with symmetry in the trophism in its two halves.
   Loss of symmetry and deviation of the tongue may indicate alterations of the central nervous system. The presence of fascicular movements, spontaneous or provoked by digital percussion, is always pathological.
3. Examine the mucosa of the right and left edges of the tongue, moving it laterally or asking the person to do so (Fig. 16.11).
4. Examine the ventral surface by asking the person to touch the hard palate with the tip of the tongue.
   Observe the characteristics of the mucosa, the superficial veins or ranulas, the frenulum and on its sides, the caruncles or exit orifices of the ducts.

Fig. 16.10 Examination of the dorsal aspect of the tongue.

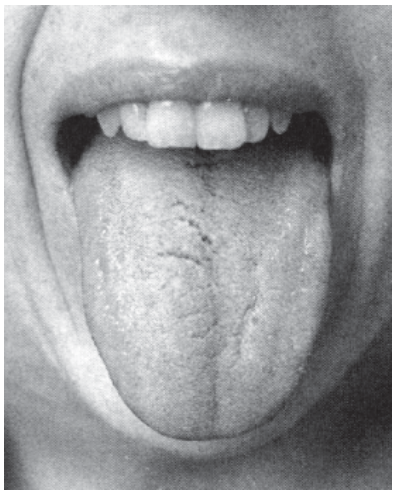


Fig. 16.11 Examination of the lateral edges of the tongue.

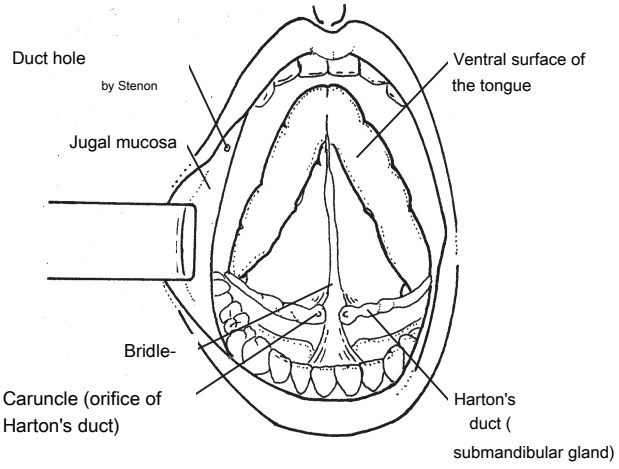


Fig. 16.12 Ventral surface of the tongue.

Wharton, which drain saliva from the submandibular glands (fig. 16.12).

The ventral surface of the tongue is smooth and shiny and the underlying veins are easily observed.

5. Inspect and palpate the mucosa of the floor of the mouth with a gloved finger and try to identify the ducts of Rivinus, by inspection, and the sublingual glands, by palpation.

Hard palate and soft palate

1. Ask the person to tilt their head back with their mouth open and examine the hard palate, illuminated directly with light or through a dental mirror.

The anterior surface of the hard palate is corrugated.

It is symmetrical with no midline openings (see Fig. 16.2).

2. Inspect the soft palate first at rest. Observe the shape and symmetry of the velum and uvula (see Fig. 16.7).

3. Ask the person to stick out their tongue and say “aaaa.”

Observe again the symmetry of the uvula and the velum, when the tongue is protruded and when the person says "aaaa."

Pair X has a branch that innervates the soft palate.

The normal function is indicated by symmetrical elevation when it says aaaa".

4. Examine the anterior crura and palatine tonsils for color, shape, and size, or describe if they are absent.

The normal mucosa that covers the tonsils is pink, similar to that of the rest of the oropharynx, and may be characterized by depressions or crypts.

Physical examination of the oropharynx

This digestive segment or middle portion of the pharynx is studied extensively in otorhinolaryngology treatises with the rest of the pharynx (nasal and laryngeal portions).

We previously explained how to inspect and palp the oropharynx, when we studied the cranial nerves. It is performed with greater or lesser ease, according to the conformation of the throat and temperament of each subject. Sometimes a depressor is not even needed and yet the pharynx is examined very well. Good lighting is always essential.

1. If the person has difficulty keeping the tongue flat, gently depress it with a depressor. Be careful not to provoke the gag reflex; Explain the procedure to the person so that they relax and feel safe.
2. Inspect the posterior wall of the pharynx and observe its mucosa, in terms of color and condition of the surface, as well as the movements of its muscles.
3. Elicit the gag reflex by touching the posterior wall of the pharynx with the depressor or mirror. Nausea with the maneuver indicates normal glossopharyngeal function (pair IX).
4. Use the dental mirror if you want to inspect the anterior aspect of the pharynx (posterior region of the uvula, tongue and posterior larynx).

EXAM AND REGISTRATION SUMMARY GUIDE

A. External structures of the oral cavity.

1. Lips and commissures: structural symmetry, deformities.

2. Dental occlusion.

3. Temporomandibular joint.

4. Chewing muscles.

5. Salivary glands.

oral cavity.

1. Mucous membranes (color and pigmentation, humidity, texture, hygiene, lesions):

a Labial mucosa.

Jugal or cheek mucosa.

C Gingiva or gum.

2. Teeth.

3. Language:

a Dorsal surface: shape, volume, color, papillae.

Structural symmetry, position, fasciculations, mobility.

Edges.

c Ventral surface: mucosa, frenulum, caruncles, lesions.

4. Floor of the mouth.

5. Palatine vault:

a Hard palate: shape, color, structural symmetry, deformities.

Soft palate: coloration, lesions. Position of the uvula. Movements and reflections.

Oropharynx.

1. Pillars.
2. Palatine tonsils.
3. Posterior wall of the pharynx: color, shape, lesions. Movements and reflections.

EXAMPLE OF RECORDING EXAMINATION RESULTS

Temporomandibular joint palpated with jaw movements: no clicks or increase in volume; restricted range of motion. Non-palpable parotids. Slight overbite, but not associated with deficiency in chewing or speaking. Labial, buccal and gingival mucosa normally colored, moist and shiny. Some teeth are missing in the lower left hemiarch. Tongue normally colored, slightly coated, moist, well papillated, located in the midline during protrusion, without fasciculations. Hard palate, normal shape, configuration and color. Uvula and soft palate rise normally with "aaaa". Tonsils: missing.

No lesions were observed or palpated on the tongue, oral mucosa or palate. Oropharynx: normal.

EXAMINATION OF THE RECTUM AND ANUS

ANATOMY FIG.

The rectum constitutes the terminal portion of the large intestine: it extends from the third sacral vertebra to the anus. In its path it crosses the posterior perineal region, limited behind by the sacrum and the coccyx, and covered by the pyramidal and ischiococcygeal muscles. The levators ani divide the rectum into two segments: the upper or pelvic segment and the lower or perineal rectum. Each of them has a different embryological origin and structure, since the lower portion originates from the cloacal membrane.

In men, the rectum is related in front to the prostate and seminal vesicles, and in women to the uterus.
Laterally it is related to the ureters, the hypogastric artery and veins, and the nerve branches of the sacral plexus.

The mucosa of the pelvic portion is smooth, pink, uniform and is covered by a columnar epithelium;
It has three well-identifiable valves or folds and a kink at its junction with the sigmoid; Its irrigation is carried out by the superior hemorrhoidal artery (branch of the inferior mesenteric) and the middle hemorrhoidal arteries (branches of the hypogastric).

The perineal rectum runs between the two ischiorectal fossae. Its light is virtual due to the constriction of the striated sphincter (external sphincter). Its mucosa represents the transition between the true mucosa and the skin, being histologically similar to the latter, but devoid of hairs and glands. Its irrigation comes from the inferior hemorrhoidal arteries (branches of the internal pudendal).

The venous system of the rectum arises from the hemorrhoidal venous plexO, which extends throughout the organ, anastomosing with the veins of the coccygeal region, the scrotum, the vulva and the inner surface of the thighs. They all drain into the hypogastric (internal iliac) vein, while the hemorrhoidal plexus forms the inferior, middle and superior hemorrhoidal veins, which are tributaries of the inferior mesenteric vein.

It follows that the pelvic rectal segment, like the rest of the colon and small intestine, ultimately drains its blood into the portal vein (through the inferior and superior mesenteric veins). Only the veins of the perineal segment of the rectum end in the hypogastric vein and, consequently, in the inferior cava. However, there are communications at the level of the rectum between the two systems (portal and cava) through the hemorrhoidal plexuses, giving rise to the formation of the inferior portocaval pedicle, which acquires its maximum development in portal hypertension syndromes (fig. 16.14). The innervation of the rectum is made up of parasympathetic fibers coming from the parasympathetic sacrum (sacral nerves SII, SIII and SIV) and by sympathetic fibers coming from the last dorsal pairs and from LI and LII.

Parasympathetic fibers are excitomotor, while sympathetic fibers decrease tone and motility.

EXPLORATION TECHNIQUES

It will be limited to the inspection of the anal region and touch rectal.

For many people, the rectal examination is an annoying and embarrassing procedure. Some men even consider it a social taboo and an attack on their manhood. The examiner must act seriously and ethically, as in any other part of the physical examination. A pleasant atmosphere must be established and with the necessary privacy, it must offer simple explanations about the procedure and the need for it, which provide security and confidence, without verbal or extraverbal manifestations that hinder the completion of the exam, violate ethics or produce iatrogenicity. You should also explain some sensations that may occur, such as the urge to defecate, when the anal canal and distal rectum are palpated. Men should be warned that they may feel an urge to ejaculate when the prostate is palpated.

Before starting the exam, it is advisable to suggest that the person go to the bathroom to empty their bladder.

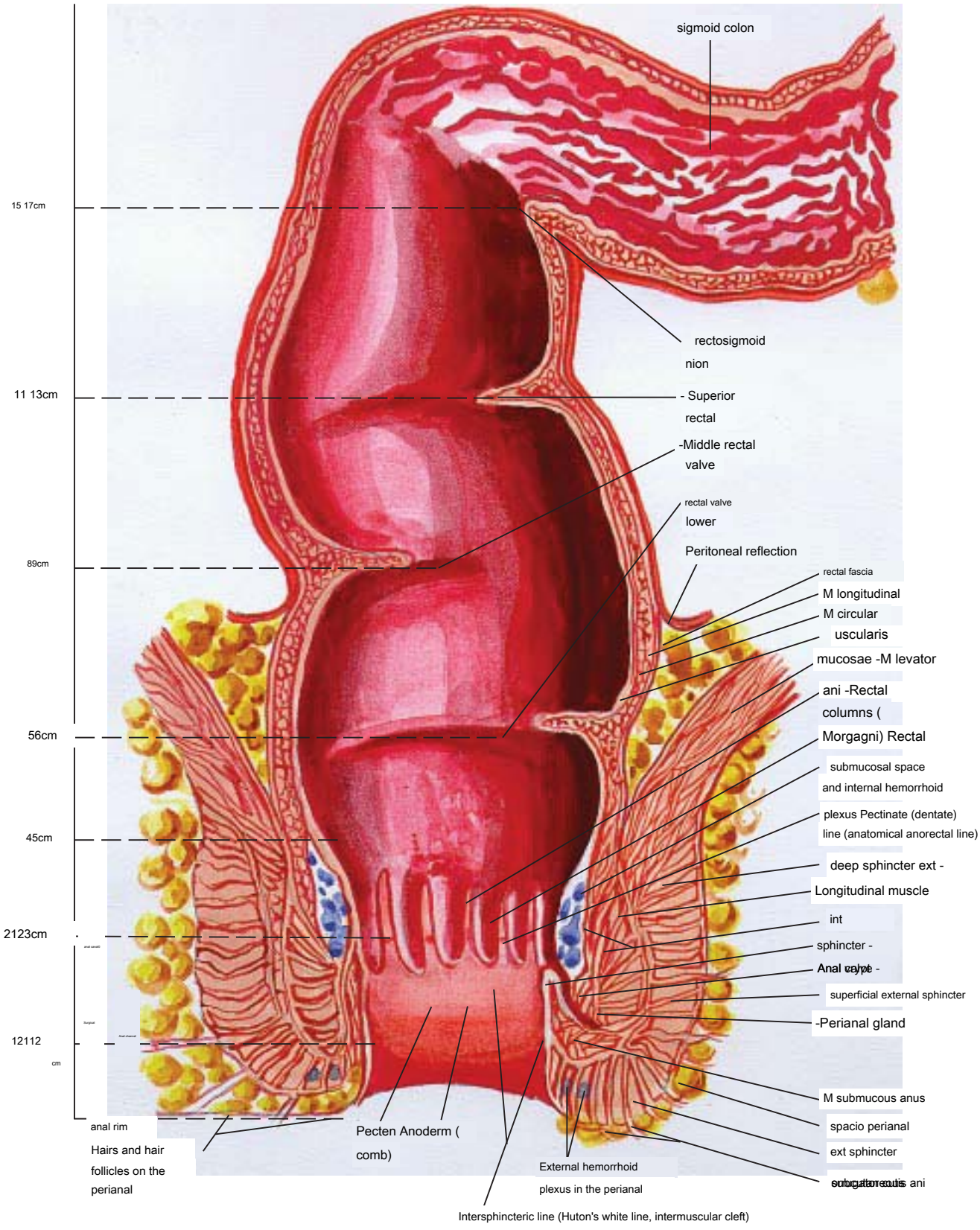


Fig. 16.13 Anatomy of the rectum and anus.

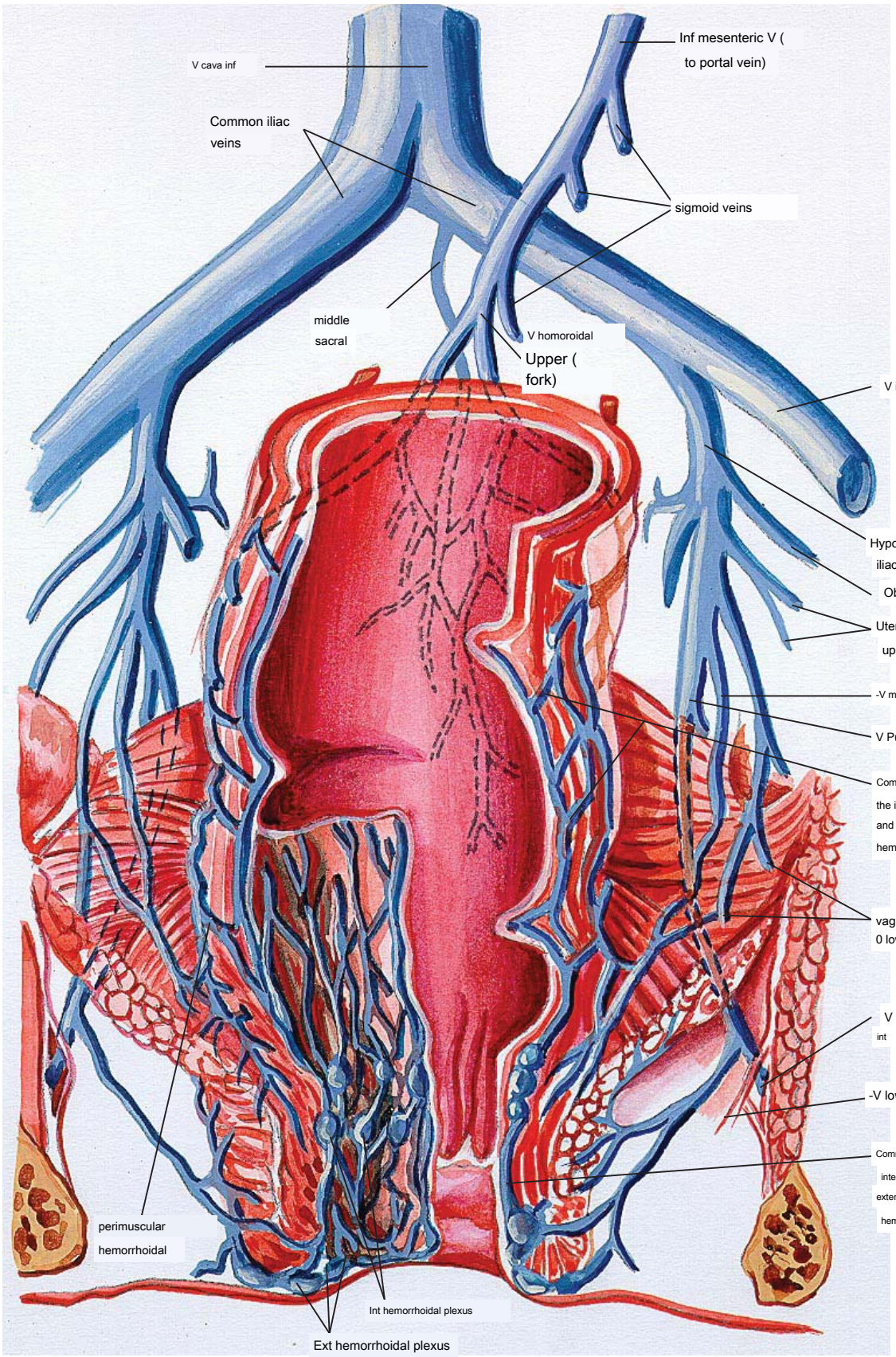


Fig. 16.14 Venous system of the rectum and anus.

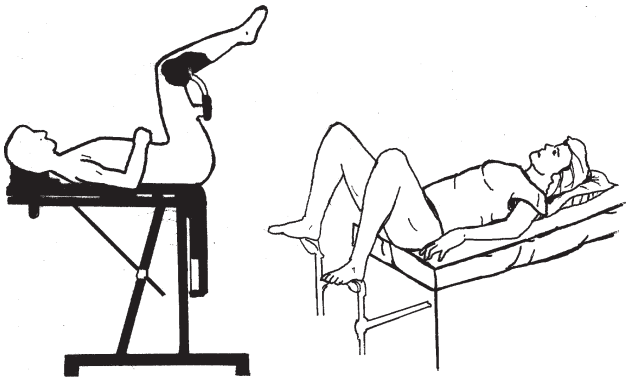


Fig. 16.15 Gynecological, dorsosacral or lithotomy position, on different tables and angles.

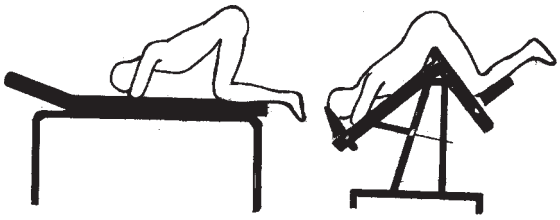
Position of the examinee. Different positions of the subject can be used, depending on the purposes of the examination and the mobility of the person.

a) Dorsosacral or gynecological position (lithotomy position). It requires the person to lie on their back, with the thighs flexed over the abdomen and the legs on the thighs, raised. This position can also be obtained by placing the heels on stirrups attached to the table. This method is generally reserved for examination in women, because the rectal examination is usually considered part of the pelvic examination of women. The lithotomy position is not ideal for men, because it does not offer the best position to palpate the prostate and, furthermore, the subject has to lift his genitals so that they do not interfere with the examination (fig. 16.15).

b) Genupectoral position. The person kneels with the hips raised and the head and shoulders resting on the examination table. The position allows optimal observation of the perineum and palpation of the prostate gland. There are tables appropriate for this position, which is the one commonly used by proctologists (fig. 16.16).

c) Standing position. The person stands at the end of the examination table and leans forward at the hips, while the upper trunk rests on a high table. Palpation of the prostate is easier in this position than in the genupectoral position, which is why it is the preferred position by urologists for its exploration (Fig. 16.17).

Fig. 16.16 Genupectoral position: a) with horizontal rigid table; b) with proctological table.



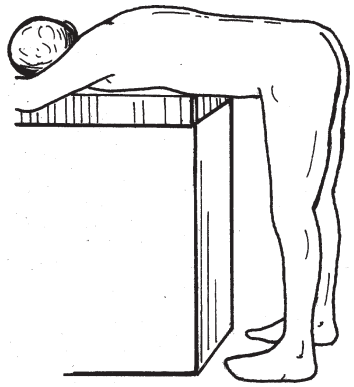


Fig. 16.17 Standing position.

d) Left lateral decubitus position (Sims position).

It is the most widely used in digestive examination and consists of the person lying on the left side, with flexion of the right lower limb, which is above, in a way that brings the knee closer to the chest, and the extension of the left lower limb, which is below (fig. 16.18).

Rectal masses, including fecal impacts, can be easily palpated in this position. The uppermost portion of the rectum may be difficult to palpate, because this position will move it away from your finger. However, it is the easiest position to assume for a person who is confined to bed.

e) Crouched position. It requires the person to crouch down, flexing the thighs and knees, while leaning slightly forward and supporting the weight and balance of the body by hugging the legs with the hands or forearms. It is a rarely used position, although rectal prolapse (protrusion of the rectal mucosa through the anus) is more easily observed with it. It also allows you to palpate a larger area

Fig. 16.18 Sims' left lateral decubitus position.

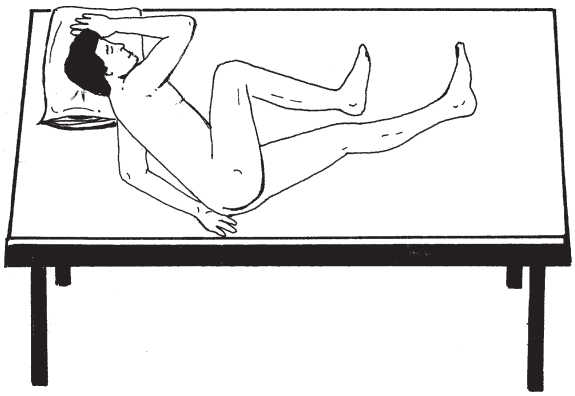


Fig. 16.19 Crouching or squatting position.

of the rectum and detect possible rectosigmoid or pelvic floor injuries (Fig. 16.19).

In the physical examination usually performed as part of a general medical history, the left lateral position is used in men and the gynecological position in women, unless a man primarily wants to perform an examination of the prostate, and then the standing position is used.

When examining a woman, perform the vulvar examination and vaginal examination before the anorectal examination to avoid contamination.

To make the person feel less uncomfortable and reduce their anxiety, modesty should be respected, covering them with a sheet from the beginning of the examination.

Except for the position, the rest of the steps of the exam are the same for men and women.

1. Place the person in a left lateral standing position. If using the lateral position, spread the glutes with your dominant hand. If using the standing position, separate the buttocks with both hands (fig. 16.20). Use a small pen flashlight (or any other artificial light source) to better assess the skin condition.

2. Examine the gluteal folds and sacrococcygeal region.

Observe the characteristics of the skin to rule out the presence of redness, secretions, bedsores or other lesions.

A bulge or hole over a bundle of hair in this area may indicate a pilonidal cyst or fistula. A pilonidal cyst is a congenital bulge in the sacrococcygeal region usually close to the upper gluteal fold, which is formed by the entrapment of a small amount of

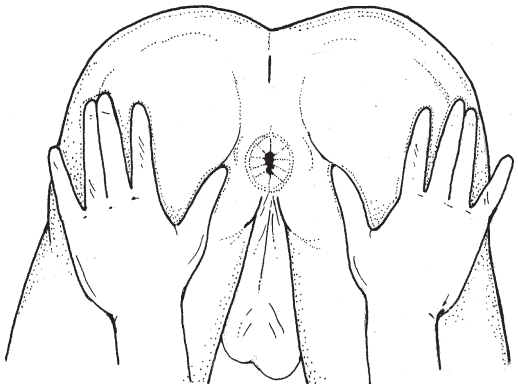


Fig. 16.20 Examination of the sacrococcygeal and anal region, standing.

hairy epithelial tissue under the skin. Hair can grow from this tissue. It is clinically important because it can become inflamed and trap debris that can become infected and lead to the formation of a pilonidal fistula.

3. Inspect the perineum, the perianal region, as well as the anus itself, observing any redness, secretions or excretions or the existence of lesions, such as scars, fissures, warts and hemorrhoids.

The skin of the perineum is continuous in color with that of the surrounding skin. The skin immediately surrounding the anus may be darker (reddish-brown), especially in boys. The surface of the skin should be free of fecal matter. The anus appears as an anteroposterior slit, which is arranged in the form of folds, which increase in number and depth parallel to the tone of the sphincter and must be carefully separated to clearly appreciate the grooves between them. The clean anus should be free of lesions or signs of skin irritation, such as erythema or rashes. The presence of fecal material may indicate poor hygiene practices. No other substances such as blood or mucus should be present.

4. Ask the person to push and then observe if there are any bulges, fissures, hemorrhoids, polyps, protruding from the anus, revealed in this maneuver.

rectal examination

Next, a rectal examination is performed. It is an exam that should never be skipped, and that Hanmilton Bayley has expressed in a very graphic phrase: "If you don't insert your finger you run the risk of screwing up."

1. Relax the external anal sphincter. Use your non-dominant hand to spread your glutes. Your dominant hand should be gloved, or the index finger with a rubber finger cot, lubricated with plain Vaseline or if necessary, any analgesic grease. Apply light pressure with the ball of your index finger against the entire anus, which will relax the sphincter and make it easier to insert the finger.

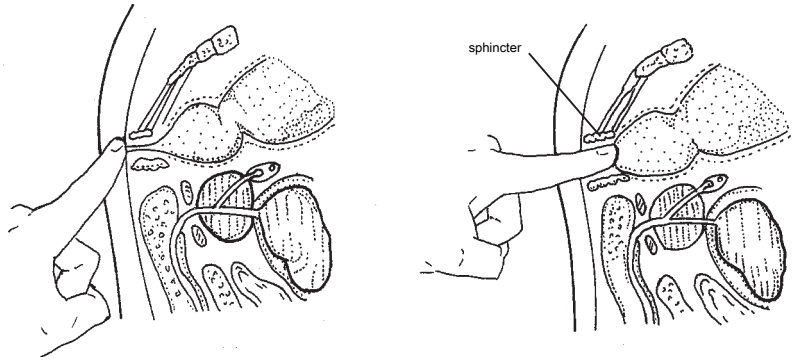


Fig. 16.21 Correct insertion of the finger into the anal canal.

now tilting it downwards, while inserting it (fig. 16.21).

Using the tip of the finger rather than the ball of the finger will cause greater pain and greater sphincter contracture.

The digital examination may be painful if the external sphincter is not relaxed before inserting the examining finger.

2. Palpate the anal canal. Insert your finger into the anal canal, pointing toward the navel, while instructing the subject to push, to further relax the sphincter.

Before palpating, ask the person to now contract the external anal sphincter, squeezing it around your finger, to assess the folds and muscle tone of the sphincter.

After the subject relaxes again, rotate your finger to palpate the entire lumen of the anal canal. Stop the test if the person experiences pain.

The external sphincter must remain closed until voluntary contraction of the levator muscles (as occurs with defecation) opens it. Good muscle tone is present if the person can voluntarily contract the sphincter around the examined finger.

swimmer

3. Palpate the levator ani muscles. Advance your examining finger into the anal canal. Palpate the levator ani muscles at the anorectal junction, in the posterolateral regions of the wall.

The levator ani muscles play an important role in bowel control. The muscles may be difficult to feel, but they should be smooth and firm.

4. Palpate the lateral and posterior walls of the rectum. Advance your exploring finger and systematically palpate the right lateral wall, the posterior wall, and the left lateral wall of the rectum. The posterior wall may be difficult to palpate because it extends further from the anal opening. Ask the person to push while you palpate the posterior wall, so that a mass higher in the rectum can be palpated (Fig. 16.22).

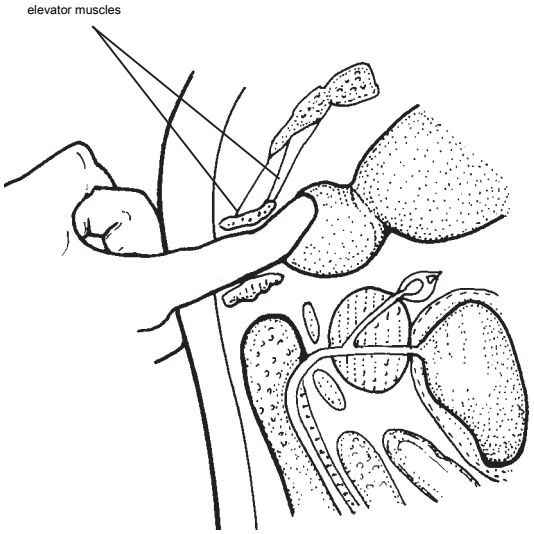
Normally the anus and rectum are permeable, checking the smoothness of their walls. They can be palpated in the rectum formed, soft stools. Hard or stone-like stools may indicate fecal impaction. If the person suffers fecal impaction, liquid stool may ooze from the rectum during the examination. If you suspect a risk that the person may defecate during the examination, place protective dressings under the buttocks beforehand.

Palpable masses are abnormal. Feces, tampons in women (felt through the anterior wall of the rectum) and the valves of Houston should not be confused with masses.

5. In men. Palpate the prostate and the anterior rectal wall. Rotate the examining finger to palpate the anterior wall (Fig. 16.23). Identify the lateral lobes and medial sulcus of the prostate gland, specifying its size, sensitivity, consistency (firm or hard) and surface (smooth or nodular).

The prostate is round, 4 cm wide by 2.5 cm long, with a palpable median groove, separating the two lobes. It should feel firm, rubbery, and free of nodules and masses. It should not be painful, although you may feel like urinating when you feel it.

Fig. 16.22 Palpation of the levator ani.



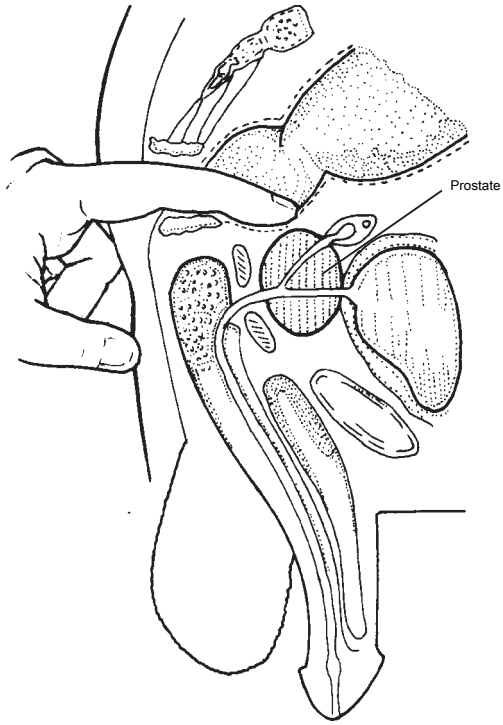


Fig. 16.23 Palpation of the anterior wall and prostate gland.

6. In women. Palpate the cervix. The anterior wall is examined in women as part of the physical examination of the pelvis (described later in this Section). Rotate the examining finger and palpate the anterior wall. Palpate the rounded tip of the neck, checking for pain or nodules. It is common that the cul-de-sac can be explored by touching the cervix. Have the woman bear down to evaluate for uterine prolapse.

Cervix. A smooth, rounded, firm mass felt through the anterior wall; not painful on palpation.

Uterus. Difficult to palpate by rectal examination, unless prolapsed.

Complete the exam

1. Slowly remove your examining finger from the rectum. Observe the color of the feces that may have adhered to the glove and take a sample for occult blood, if what is necessary to perform the test is available. Be careful not to contaminate the perineum with fecal matter that may be stuck to the gloves.
2. When you finish the exam, offer the person an appropriate paper or cloth to wipe with, or clean the perineum yourself.
3. Thank them for their cooperation.
4. Wash your hands. Even if you wear gloves during the exam, good hand washing is essential after completing the exam.

AND USE OF THE DELEXAMEN

REGISTRATION

Folds, glutes, sacrococcygeal, perianal and anal regions, free of lesions. Clean anus, with normal folds, no external hemorrhoids or other lesions.

Rectal examination: external sphincter in good tone. Rectum occupied by a small amount of soft stool, smooth walls, no internal hemorrhoids, no masses or pain. Prostate smooth, firm, non-painful and without masses; size 4 x 2 cm. The glove comes out stained with normal colored feces, not blood or other secretions.

PHYSICAL EXAMINATION OF THE LYMPH SYSTEM

AND OF THE BA O

17

EXPLORATION OF THE LYMPH SYSTEM

The lymphatic system is made up of lymphatic vessels and nodes.
It intervenes in adult hematopoiesis, originating, first, lymphocytes, and second, monocytes and lymphatic plasma cells.

Lymph is produced by the tissues and collected by the lymphatic channels that join to form the lymphatic vessels and passes through the intercalated lymph nodes, where it is supplied with abundant lymphocytes and is ultimately poured into the blood through the thoracic duct and the lymphatic trunk that empties into the right yugosubclavian venous angle.

About 2 L of lymph enters the blood every 24 hours.

With more functional than anatomical criteria, the lymphoepithelial organs of Waldeyer's peripharyngeal ring (tonsils), the thymus, the follicles and Peyer's patches of the intestinal submucosa and even the spleen, whose structure allows it to be considered a large intra-abdominal lymph node, are considered to belong to the lymphatic system. All of these formations are involved in lymphocytopoiesis and therefore must be considered linked to the lymphatic system.

There are more than two hundred lymph nodes in the body and they are located, above all, near the great vessels and on the flexing surfaces.

The lymph node chains or groups may have a deep location, for example, in the mediastinum or abdomen, which cannot be accessed by the usual physical examination, or a superficial location, which allows them to be physically explored.

Superficial lymph nodes are located in the head, neck, supraclavicular and subclavicular areas, axillae, epitrochlear areas, inguinal regions, and in the popliteal fossae (Fig. 17.1).

Because lymph nodes are distributed throughout the body, examination of the lymphatic system can be performed during the physical examination of each region. For this reason, the examination of the lymph nodes of the head and neck was addressed after the examination of the head and the nodes of the breast and axillary region, when the examination of the tốthorax and breasts was studied, all in the chapter on "Regional physical examination", and the examination of the nodes of the inguinal region will be detailed in Chapter 19, corresponding to the reproductive system, so we refer the reader, to these chapters.

As we already know, the exploration of lymph nodes is carried out by inspection and palpation, but the greatest elements are provided by palpation.

Normal lymph nodes are usually not visible or palpable.

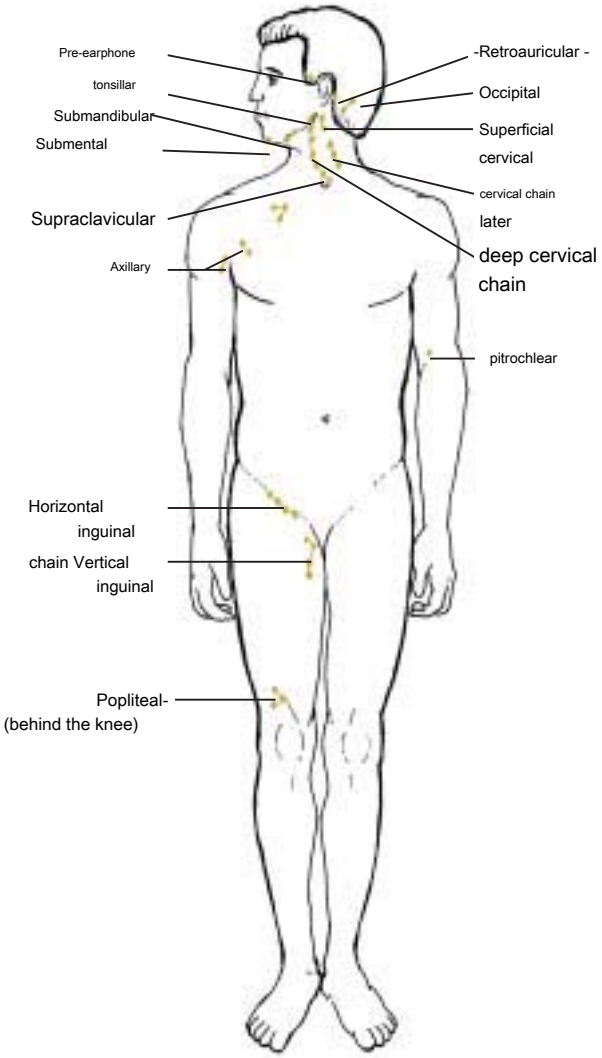
The superficial lymphatic system is examined for abnormalities such as enlarged lymph nodes, tender nodes, lymphedema, and lymphangitis.

Enlarged lymph nodes can basically represent a benign deviation from normal, a current (acute or chronic) or past local or generalized inflammation, or a neoplasm.

Lymph nodes may be painful on palpation in association with acute inflammatory processes. Lymphadenitis refers to inflammation of the lymph nodes.

Lymphedema is the increase in tissue volume due to the retrograde movement of lymph into the space.

Fig. 17.1 Sites of the ganglionic chains



interstitial from the lymphatic vessels, produced by the obstruction of flow through their vessels.

Lymphangitis is an inflammatory process along the path of a lymphatic vessel, manifested by a red streak on the skin. It is caused by bacterial spread through lymphatic vessels to the nodes, so it is usually accompanied by lymphadenitis.

If lymph node abnormalities are detected, you should carefully examine the area of the body that drains to the affected lymph node chain, looking for signs of inflammation, infection, enlargement, or lesions.

EXPLORATION TECHNIQUES

Inspection. Location of a visible lymph node, presence of increased volume or red lines or paths.

Palpation. There is variability among individuals in the number and location of lymph nodes in a particular area. Therefore, you must palpate the entire region where a lymphatic chain can be located.

Enlarged lymph nodes are more easily detected by light palpation than by deep palpation. While palpating, ask the person to report any discomfort or pain.

Palpable lymph nodes must be distinguished from underlying tissue such as muscle.

Remember that palpable lymph nodes are described in terms of location, size (mm or cm), shape, consistency, delineation, mobility, and tenderness or pain. Unlike the surrounding tissues, a swollen lymph node can usually move up, down, and sideways between the examiner's fingers.

If you detect any lymph nodes, remember to ask when it was first noticed, either by the subject or by another person, or if it had not been noticed before.

1. Examine the lymph nodes of the head and neck, the supra- and infraclavicular ones, and then those of the axillary regions, whose distributions and exploratory techniques were already described in Chapter 4.
2. Examine the epitrochlear lymph nodes. The epitrochlear nodes are located just above the medial epicondyle of the humerus. To palpate them, flex the subject's elbow 90° and palpate above the epicondyle, in the indentation created by the biceps and triceps muscles.
3. Examine the lymph nodes of the inguinal regions. The superficial inguinal lymph nodes should be palpated with the subject supine, with the knees slightly bent. Palpate

looking for the horizontal superficial inguinal chain, along the inguinal ligament. Palpate for the vertical superficial inguinal chain, just medial to the femoral vein.

4. Examine the lymph nodes in both popliteal fossae. Palpels in the posterior fossa of the knee. Palpation is facilitated by placing the knee in a slightly flexed position.

PHYSICAL EXPLORATION OF THE BA O

The physical examination procedures of the spleen are: inspection, palpation and percussion. Auscultation is not very useful, therefore we will only describe the first three mentioned.

INSPECTION

Normally the spleen is not visible, so the inspection is carried out only to rule out the presence of considerable enlargements of the spleen, which bulge the abdomen in an oblique direction, from the left hypochondrium, towards the right iliac fossa.

PALPATION

A normal spleen is never palpable. So the exploratory technique is performed to rule out splenomegaly or splenic ptosis. Usually, the spleen must have grown three times its normal size before it is palpable.

A. Perform palpation in supine position.

1. Ask the person to stand with their legs extended and arms at their sides, and to breathe widely and calmly. The position will be more comfortable with a not very high pillow on which to rest your head.

2. Stand, or better yet, sit to the right of the subject; Take the last ribs (IX and

The spleen is perceived at the end of inspiration, during which it descends following a line that joins the apex of the left armpit with the midpoint of Poupart's arch.

B. Perform palpation in a diagonal position (Schuster position) .

1. Place the subject with the thorax in an oblique position, intermediate between the dorsal and right lateral decubitus position; pelvis and legs in complete right position; the right lower limb remains extended, while the left side flexes, the muscle

Fig. 17.2 Palpation of the spleen in dorsal recumbency.

it on the pelvis and the leg on the thigh. The head is supported by a pillow, and place another small pillow behind the left scapular region, to prevent the person from using force to maintain the position. The left arm should be in front of the chest resting on the bed without the shoulder rising.

This precaution is very important, because if it is carried upwards above or in front of the head as is commonly done, when the shoulder is raised, the rib cage will rise, the abdominal muscles will be stretched and palpation will be difficult.

2. You can sit on the right or stand on the left. In the first case, perform bimanual palpation as described for the supine position: the left hand to support the costal wall and the right to palpate. If you are standing on the left, try to cover the spleen that protrudes from the costal margin with your hooked fingers: this is the so-called hooking maneuver (fig. 17.3).

C. The Ziemmsen semi-sitting position is used to achieve greater drooping of the spleen.

1. Lie the subject on three or four pillows, in an oblique position, intermediate between the supine position and the sitting position.

Fig. 17.3 Palpation of the spleen. Hitch maneuver.

2. You can be seated on the right and palpate according to the rules indicated regarding bimanual palpation in supine and right lateral decubitus.

D. Llanio's position.

It is based on studies carried out through laparoscopy, during which the spleen can be directly visualized and its position and displacement with changes in position. Its author recommends placing the patient, with the body in a complete oblique or right lateral position, on a table with an inclination of approximately 45°, so that the head and thorax are elevated and the abdomen and lower limbs are lowered, that is, in the inverted Trendelenburg position.

In this position the spleen descends and leaves its position behind the ribs, as can be easily verified during laparoscopy.

If an examination table is not available, pillows can be used to achieve the indicated position, or the head of the stretcher can be raised with a wedge or

bed.

Of course, all these maneuvers are necessary when the spleen is not very enlarged, because if so, it can be easily palpated even in the supine position.

Palpatory recognition of the spleen

The spleen has the following characteristics:

1. Location below the costal margin between the IX and XI ribs.

2. Superficial situation immediately below the abdominal wall.

3. Anterior edge with incisions or notches.

4. Respiratory mobility wide and diagonally towards the navel.

The technical errors most frequently committed by students when beginning palpation of the spleen are:

1. Not previously performing a palpatory examination of the abdominal region. This single maneuver allows us to discover very large spleens. If it is not performed previously, even large splenomegaly can go unnoticed or be confused.
2. Start palpation above the costal margin instead of starting lower.
3. Make circular movements with the tips of your fingers, almost as if you were massaging the skin and cellular tissue (a very common mistake).
4. Perform very deep palpation, forgetting that the spleen is very superficial.
5. On the contrary, perform very superficial palpation without depressing the muscular wall.
6. When the muscular wall is somewhat tense, confuse it with the lower pole of the spleen.
7. When inserting the tips of your fingers under the costal arch, palpate the muscular fingerings and believe you are touching the splenic pole. It is enough to run your fingers along and under the costal margin to get out of the error, because the muscle bundles continue to be touched.
8. Make the subject breathe deeply, without having performed prior palpation with calm breathing, which leaves the belly wall soft and allows the characteristics of the organ to be comfortably recognized, when it is palpable even during expiration.
9. Start palpation of the spleen in Schuster's diagonal position, instead of starting in the supine position.

PERCUSSION

It is valuable in revealing discreet and unnoticed splenomegaly on palpation. It is the only means of appreciation when the organ is not yet parietoabdominal.

The spleen is located between the IX and XI ribs, along the The left lung is interposed in its upper third and therefore evades percussion. Splenic dullness corresponds to the anteroinferior two-thirds of the organ, usually directly attached to the thoracic wall.

To percuss the spleen:

1. Place the examinee in the right lateral decubitus position. This position is the best, because with it the air chamber, almost always present, even with the stomach full of food, is arranged in a way that facilitates the demarcation of the lower splenic end.
2. Strike between the middle and posterior axillary lines, from top to bottom and very gently. Drumming at ordinary intensity, too energetic, is a mistake made by most explorers. Between the VIII and IX ribs the splenic dullness is obtained, which, as we have previously pointed out, corresponds to the union of the upper third of the organ with its lower two thirds.

3. From the point at which splenic dullness was obtained, follow a descending line, parallel to the course of the ribs, until finding a clear tympanism that serves to delimit its anteroinferior pole.

On the anterior side, the splenic dullness should not exceed the anterior axillary line or the costoarticular line (the one that joins the sternoclavicular joint and the lowest point of the costal arch) and from below it should be vertically about 5 cm away from it. If the dullness of the spleen measures more than 7 cm in height in the direction of the body axis, in the adult of average size it should already be considered pathological.

18

EXPLORATION TECHNIQUES

The urinary system is explored by:

- Inspection.
- Palpation.
- Percussion.
– Auscultation.

INSPECTION

In general terms, this method provides little data; However, we should point out that sometimes inspection can reveal the presence of tumors on one or both sides of the abdomen (hypochondria and flanks), an expression of unilateral or bilateral renal enlargement.

PALPATION

Normally the kidneys are not palpable. There are classic maneuvers that are useful mainly to demonstrate the physiological condition, that is, the absence of palpable kidneys or their discrete growth or decrease. These maneuvers are:

- Guyon bimanual procedure.
- Kidney rally.
- Glenard maneuver.
-Goelet method.
- Renoureteral painful points.

Bimanual Guyon procedure

The subject should remain lying face up, silent, totally relaxed, and the doctor should sit on the same side of the kidney being examined. Place your left hand in the examinee's lumbar region, if you are trying to palpate the right kidney, with the tips of the fingers 566 cm from the midline, so that it is over the renal fossa and exerts moderate and constant pressure against it (fig. 18.1).

The right hand is placed on the anterior wall, below the costal margin, on the external limit of the rectus femoris on that side. The fingers should remain in a plane parallel to the abdominal wall, the doctor applies pressure with the tip of the fingers, not the tip, and holds them.

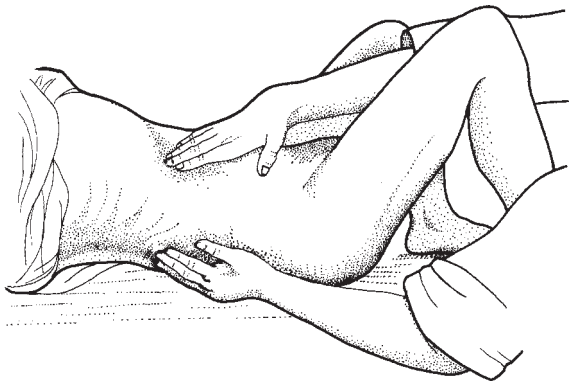


Fig. 18.1 Palpation of the kidney. Guyon bimanual procedure.

They are erect, making the necessary movements at the expense of the metacarpophalangeal joint; The right hand should go deeper towards the posterior wall, pushing the fingers only when inhaling until the kidney on that side is palpated, when it is descended or increased.

Kidney rally

Guyon himself described the rally maneuver, which consists of producing with the tips of the fingers of the left (posterior) hand, dry and repeated impulses on the posterior wall, keeping the right (anterior) hand flat in the place already described (fig. 18.2). When there is a palpable kidney or other tumor that makes lumbar contact, the right (anterior) hand perceives a sensation of balling.

When the maneuver is positive, the right hand perceives a soft intermittent shock in the abdomen that corresponds to the kidney that bounces around in its gaseous atmosphere, due to the impulse caused by the hand located in the posterior or lumbar region.

For a rally to be genuinely renal, several precautions must be taken:

a) The lumbar impulse must be carried out at the costomuscular point, since only the kidney bounces in that area.

b) The shock perceived by the hand on the abdomen must be intermittent, that is, not maintained during the entire time in which the other hand pushes from the lumbar region, such a fact is not a true rally, but rather a mass excursion, typical of any tumor that can be reached from the back.

c) The entire maneuver must be performed at the level of the midclavicular line, because if it is done outside of it, the hand on the abdomen perceives a false rally created by the hand located in the lumbar region through the scarce cutaneous-muscular mass of the flank, an illusion that is all the more possible the more flaccid the subject's abdomen is. In case of doubt, this lateral transmission of the lumbar impulse can be interrupted by placing the thumb of the hand located in the lumbar region on the flank.

For the left kidney, the procedure is the same:

Only the respective positions of the right and left hands are reversed, that is, located in the lumbar and abdominal regions, and the doctor stands to the left of the person to be examined. In general, the left kidney is more difficult to palpate than the right, because it is higher than the right.

Glenard maneuver

It was described by this author to discover ptosis and classify them into degrees. The examinee and the doctor are positioned in the same way as in the previous procedure. Three times are described: stalking, capture and escape.

Stalking. The left hand is placed (if the intention is to palpate the right side) so that the upper edge of the middle finger is below and parallel to the twelfth rib, its end reaching the limit with the right lumbosacral mass. The thumb is left in front, opposite the middle finger, forming a kind of pincer with it (fig. 18.3).

The right hand, with the fingers - except the thumb - aligned and directed upwards and outwards on the flank

Fig. 18.2 Palpation of the kidney. Rally maneuver.

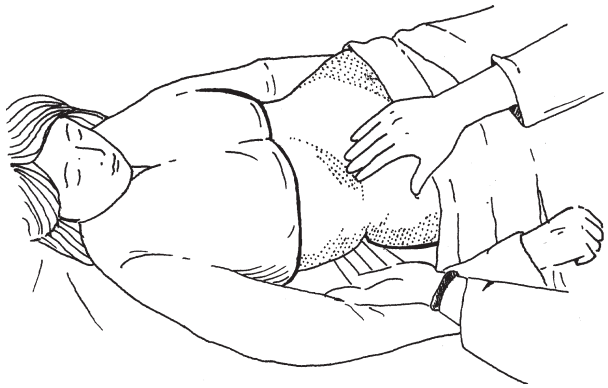
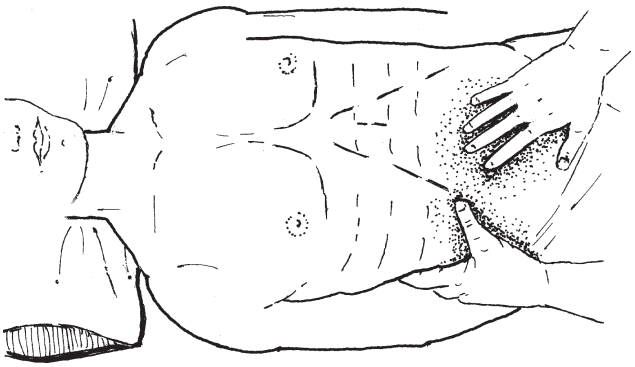


Fig. 18.3 Palpation of the kidney. Glenard maneuver (stalking).



right, it exerts pressure on successive points, from bottom to top, along a line that goes from the xiphoid process to the middle of the inguinal fold. The purpose of this hand is to oppose the lateral displacement of the kidney, to prevent its pole from falling inwards and upwards, and to bring it outwards, so that it can be captured between the clamp formed with the left hand.

Capture. During inspiration, the palpable kidney passes over the thumb clamp, which captures it during post-inspiratory apnea. If this does not occur, there is no true ptosis.

Exhaust. During expiration, the kidney escapes from the digital clamp, moving upward or remaining fixed by the clamp, which is common in renal ptosis.

There are other palpatory methods in an upright position that tend to facilitate the descent of the kidneys. Among these methods we have Goelet's.

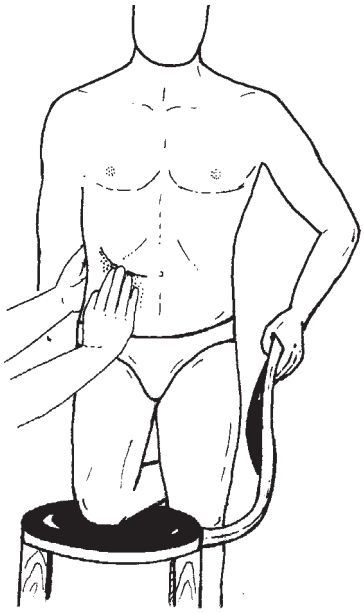
Goelet method

The examinee stands on the lower limb opposite to the side to be palpated, and the other limb rests, flexed, on a chair, in order to relax the abdomen. The procedure is bimanual, with one hand on the lumbar area and the other on the abdomen, watching for inspiration of the kidney (fig. 18.4).

Renal ureteral pain points

The points that we point out below are intended to awaken, through palpation, pain at different levels of the nerve path to which kidney pain usually refers, which ultimately come from the ulcers.

Fig. 18.4 Palpation of the kidney. Goelet method.



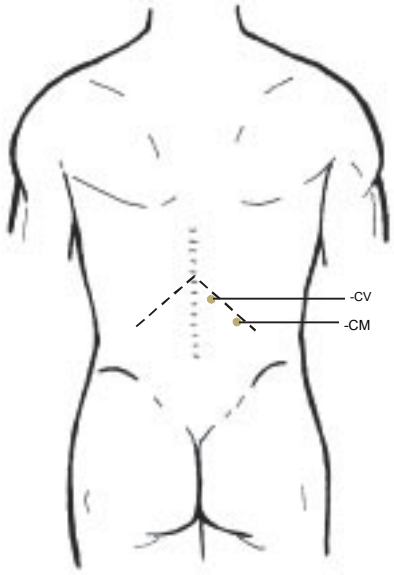


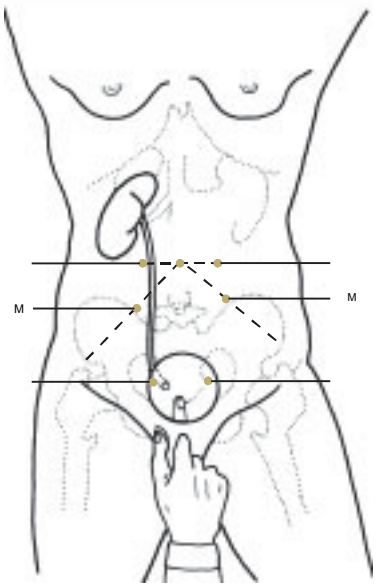
Fig. 18.5 Posterior renoureteral tender points: CV, costovertebral; CM, costomuscular.

thymus dorsal and first lumbar roots. Their inconsistency in nephropathies and their occasional presence in other conditions mean that they retain only a relative value and that a few maintain a certain validity.

The painful points themselves are:

1. Posterior (fig. 18.5):
   a) Costovertebral.
   b) Costomuscular.
2. Previous (fig. 18.6):
   a) Subcostal.
   b) Upper ureteral or pelviureteral.

Fig. 18.6 Anterior renoureteral tender points: US, upper ureteral; UM, middle ureteral; UI, lower ureteral.



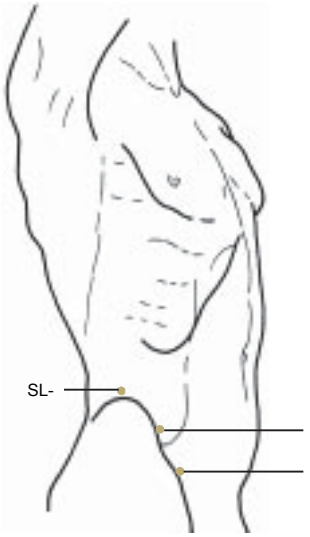


Fig. 18.7 Remote painful points: SIL, Lateral Suprailiac; SIE, supraintraspinatus; I, inguinal.

c) Middle ureteral.

d) Lower ureteral or juxtavesical.

3. At a distance (fig. 18.7):

a) Supraintraspinatus.

b) Inguinal.

c) Lateral suprailiac.

Let's see below how some of these points are determined:

Costovertebral. It is sought in the angle formed by the lower edge of the XII rib with the vertebral column.
It corresponds to the exit, through the foramen of conjunction, of the twelfth intercostal nerve.

Costomuscular. It is explored at the junction of the lower edge of the XII rib with the outer edge of the spinal muscle mass. It corresponds to the posterior perforating branch of the twelfth intercostal at the point where it becomes superficial.

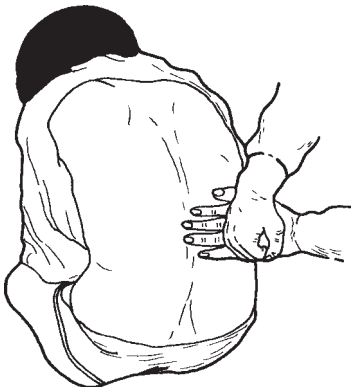


Fig. 18.8 Percussive fist maneuver.

Superior ureteral or pelviureteral. It is sought at the level of the umbilical line, at its intersection with the external border of the rectus femoris. It corresponds to the ureteropelvic junction. It is usually painful in pyelonephritis, renoureteral lithiasis, etc.

Middle ureteral. It is sought at the junction of the biiliac line with a vertical line raised from the pubic spine.

Lower ureteral. This point, which corresponds to the entrance of the ureter into the bladder, is explored by rectal or vaginal examination.

PERCUSSION

Finger percussion, or more commonly fist percussion, at the level of the lumbar fossa, awakens or intensifies lumbar pain of capsular origin (Fig. 18.8).

AUSCULTATION

Auscultation of the lumbar regions and flanks has acquired renewed interest in relation to the etiological investigation of arterial hypertension.

The search for murmurs should be done with the subject in a lateral decubitus position, with the thighs flexed over the abdomen (to relax the abdominal wall well), sinking the stethoscope deeply into the region being listened to and in a quiet environment.

EXPLORATION OF THE MALE REPRODUCTIVE SYSTEM

19

ANATOMY ISTOLOGY AND PHYSIOLOGY OF THE MALE GENITAL ORGANS

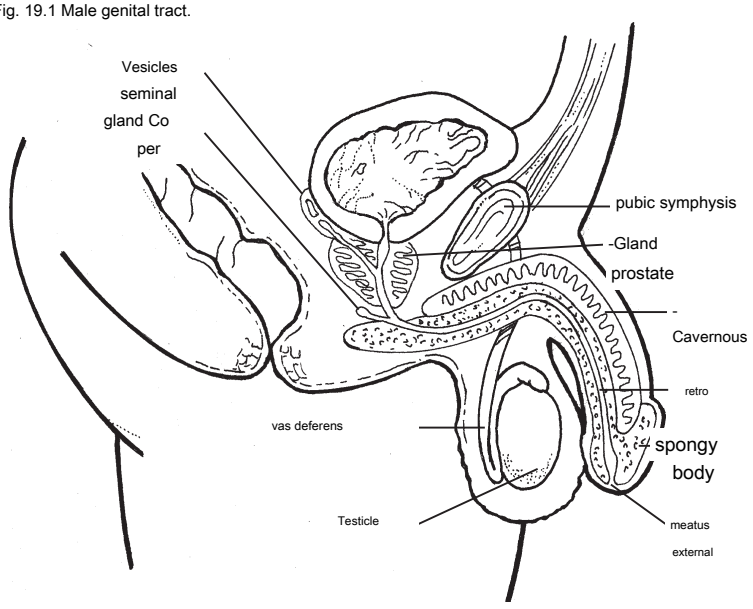
The male genital apparatus is essentially made up of two parts:
a glandular organ (the testicles) in charge of producing fertilizing fluid (sperm), and the ducts intended to transport this fluid to the woman's copulatrous sac, which constitute what we call the spermatic tract. To these essential parts we must add: the bags or scrotum; a copulatory organ (the penis) and a number of glands (prostate and Cowper's glands) (fig. 19.1).

TESTICLES

In men, the testicles are paired organs, ellipsoid or ovoid in shape, suspended in the scrotum by the spermatic cords. They are made up of vessels, nerves and " vas deferens" or excretory ducts (fig. 19.2).

The total weight of the two glands is 21-28 g, they measure 4-5 cm long, 2.5 cm wide and 3 cm in anteroposterior diameter.

Fig. 19.1 Male genital tract.



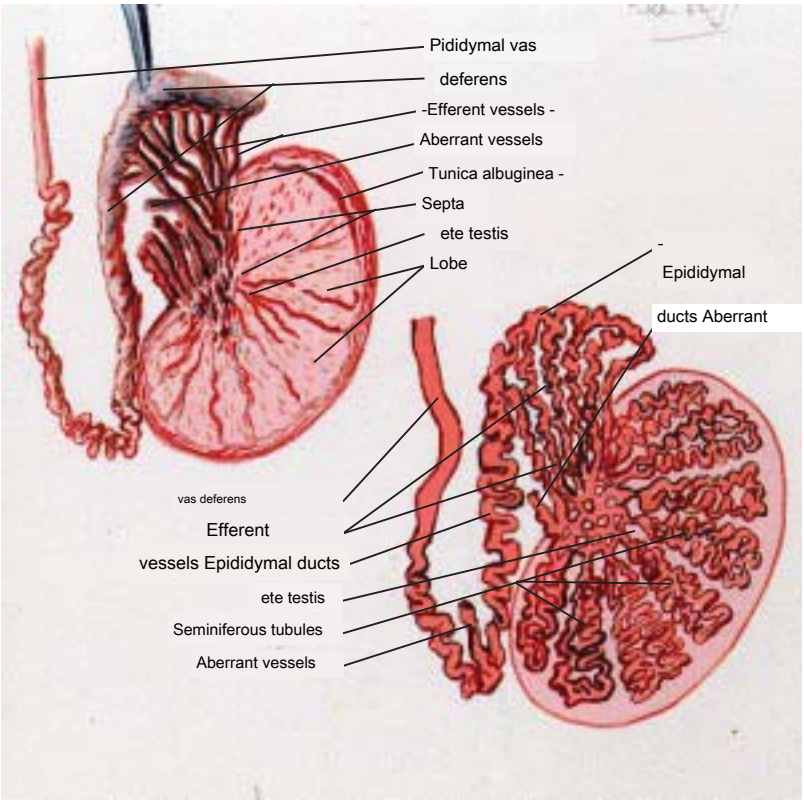


Fig. 19.2 Anatomy of the testicle, epididymis and vas deferens.

The surface of the testicle is covered by the visceral sheet of the tunica vaginalis, which is part of the vaginal sac of the peritoneum. Below this layer is the tunica albuginea, which is the fibrous capsule of the testicle. At the posterior edge of this, the capsule invades the gland, forming a septum or mediastinum testis. From this septum, other septa detach in a radiated form that divide the testicle into lobules in number of 250-400. Each of these lobules contains 3-4 seminiferous tubes.

The male gonads or testicles, like the female ones, fulfill two fundamental functions in the body: they are the organs of reproduction and they secrete active hormones that influence the development, maturation and homeostasis of men.

The testicles, housed in the scrotal bags, fulfill their secretory function through the Leydig cells, and the reproductive function or spermatogenesis, through the epithelium of the seminiferous tubules, which, starting from the spermatogonia and in six progressive stages, reaches the production of mature spermatozoa, which it renews every 16 days, while the total duration of the evolution of the spermatogonia requires a longer interval, about 74 days (fig. 19.3). The third cellular element, the Sertoli cells, without their own capacity to produce hormonal substances, act as nutrient elements for the less mature types of sperm.

Both the secretion of testosterone by the Leydig cells and spermatogenesis are under the control of the same pituitary hormones that we mentioned. oars for the ovary. FSH stimulates the germinal epithelium to produce spermatogenesis and maintains Sertoli cell trophism; while LH, or interstitial cell-stimulating hormone, stimulates the biosynthesis of androgens and estrogens in Leydig cells. These androgenic substances, while stimulating the growth of the seminiferous tubules, give men their secondary sexual characteristics, but fundamentally they stimulate spermatogenesis.

Physiology

Within the process of androgenic biosynthesis, testosterone (the fundamental androgenic steroid of the testicle) is formed in this organ by two biosynthetic pathways: first from delta-5-pregnenolone, which has as an intermediate step 17-alpha-hydroxy-pregnenolone and delta-4-androstenedione dehydroepiandrosterone on the one hand, and second from progesterone and 17-alpha-hydroxyprogesterone and delta-4-androstenedione. In both cases, the steroid prior to testosterone is delta-4-androstenedione. The latter and dehydroepiandrosterone are also secreted in smaller quantities by the testicle and also have a lower androgenic action (fig. 19.4).

Daily the testicle produces around 7 mg of testosterone; This production appears to have a nycthemeral rhythm. Testosterone also has a loose binding to a plasma carrier protein and is metabolized in the liver, creating degradation products, which

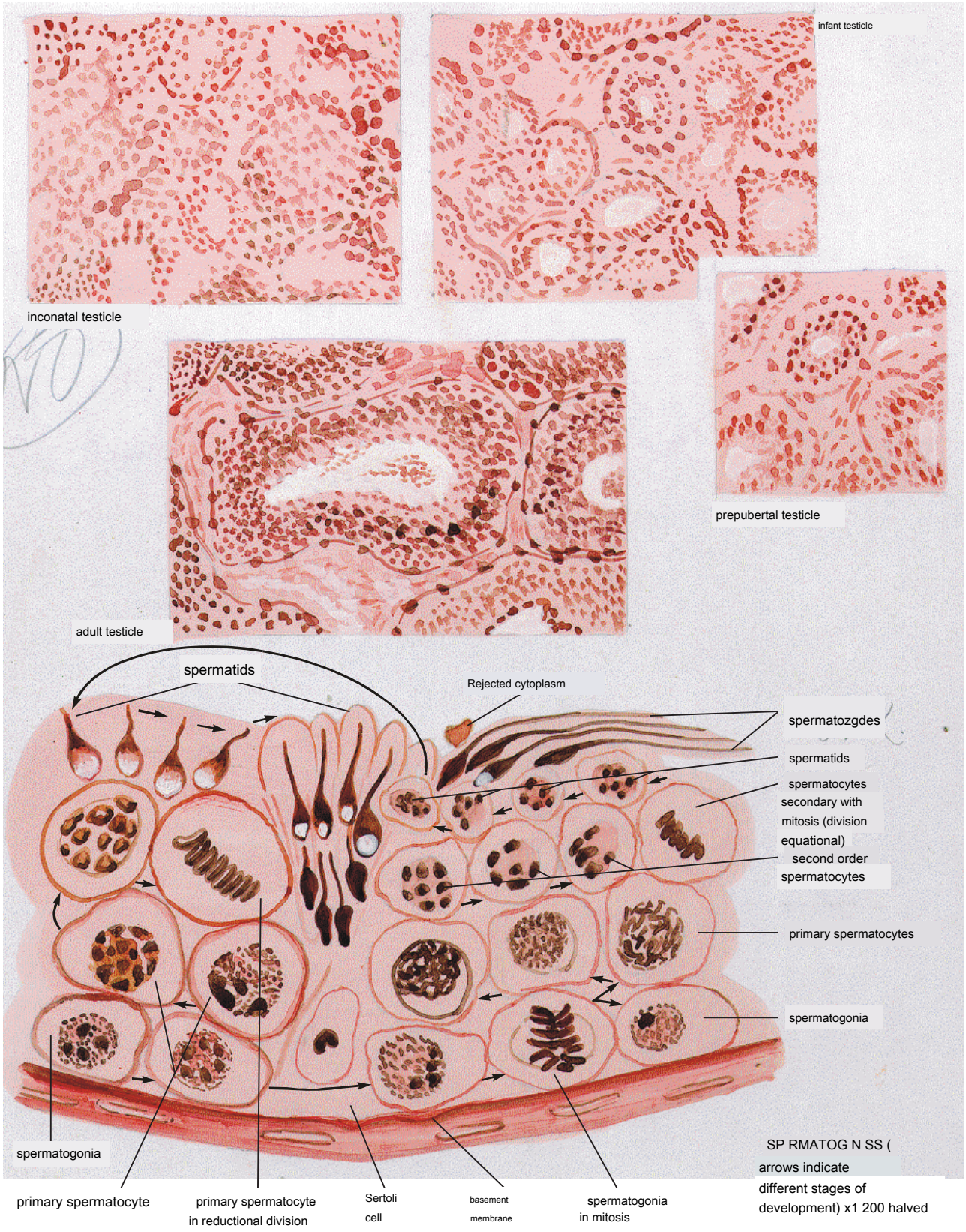


Fig. 19.3 Spermatogenesis.

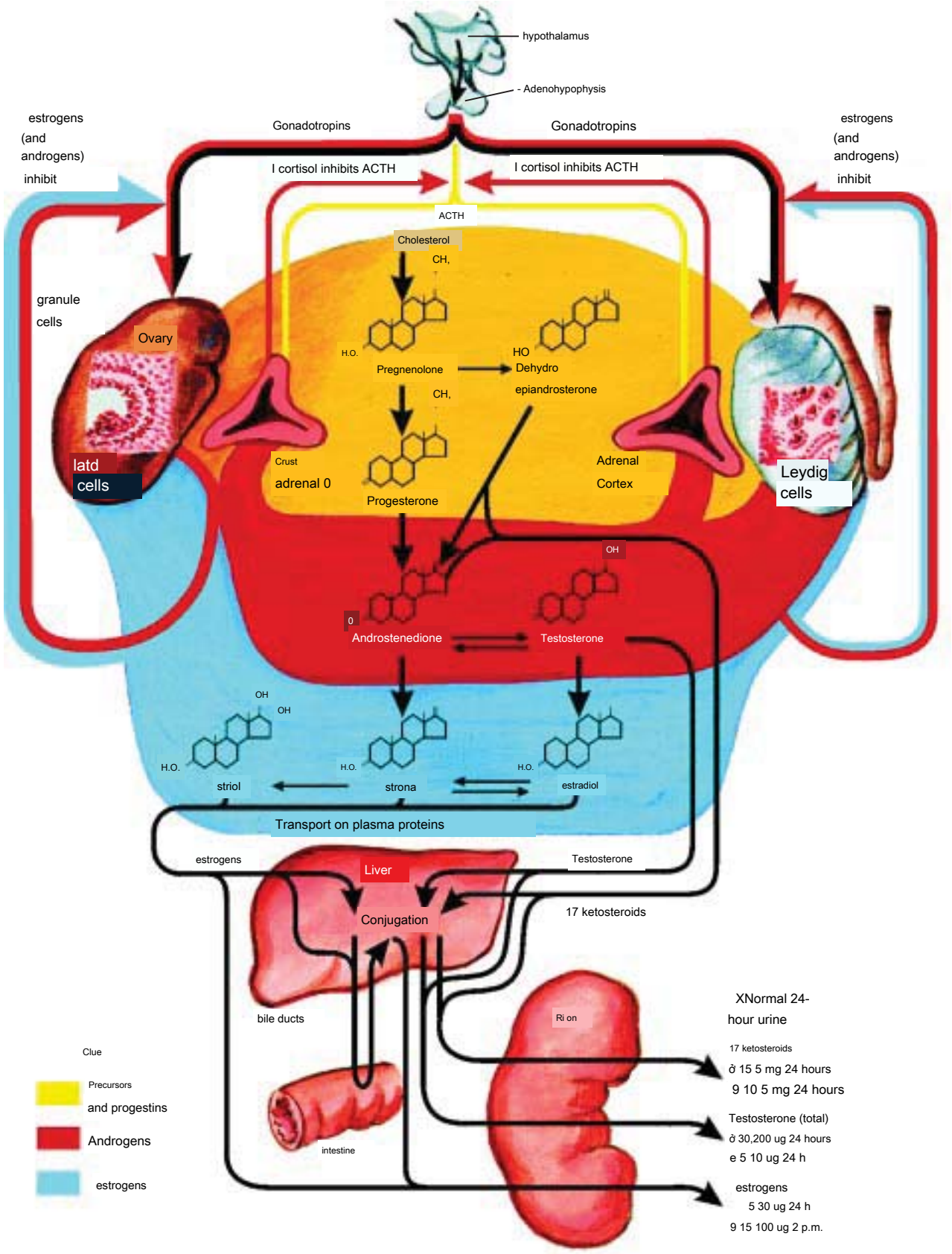


Fig. 19.4 Synthesis of testosterone and estrogens in the testis.

Conjugated with glucuronic and sulfuric acid, they are eliminated in the urine in the form of 17-ketosteroids. The determination of urinary 17-ketosteroids, which involves the assessment primarily of androgenic substances and secondarily of other inactive substances produced by the adrenal gland and the testicle, constitutes only 30% of the daily production of testicular androgens, while the other 70% is formed by the adrenal gland. For that reason, it is worth repeating here that not all androgens are eliminated as 17-ketosteroids.

The conversion of testosterone into estrogenic substances takes place in the testicle and is of great importance.

Testosterone, through its conversion into dehydrotestosterone as a typical male sex hormone, causes at the time of puberty the growth of pubic hair, an increase in the size of the penis and the pigmentation of it and the scrotum, the appearance of a beard and mustache, "the rapid growth of puberty", the growth of the prostate and seminal vesicles as well as a significant increase in protein anabolism, with retention of nitrogen, potassium and calcium.

Androgens promote increased protein synthesis and amino acid retention. A male climacteric has also been described in men, with symptoms similar to those of the female (hot flashes, decreased libido and potency, and mental depressive syndrome).

The absence of androgen secretion by the testicle, as in the ovary, may have a testicular or pituitary origin, from which it can be inferred that this decrease, generally classified as hypogonadism, may be due to pituitary causes or deficiencies specific to the testicle.

SCROTAL BAG FIG.

The testicles, once they have migrated from the abdomen, are contained within a system of concentric covers that are called bags.

The bags appear as a voluminous, odd prominence located in the midline, in front of the symphysis, in the space left in front of the thighs, and held by its upper end, it is free in the rest. This ovoid-shaped eminence measures, in the adult, 6 cm high by 5 cm wide and 4 cm thick.

Small and consistent in the child, they are flaccid and hanging in the elderly. On its anterior surface there is a median groove, at the bottom of which there is a raphe (an indication of the primitive duplicity of the organ).

SPERM TIC PATHWAYS

The sperm pass from the seminiferous tubes to the epididymis and upon leaving this, the sperm successively travels through the vas deferens, the seminal vesicle and the

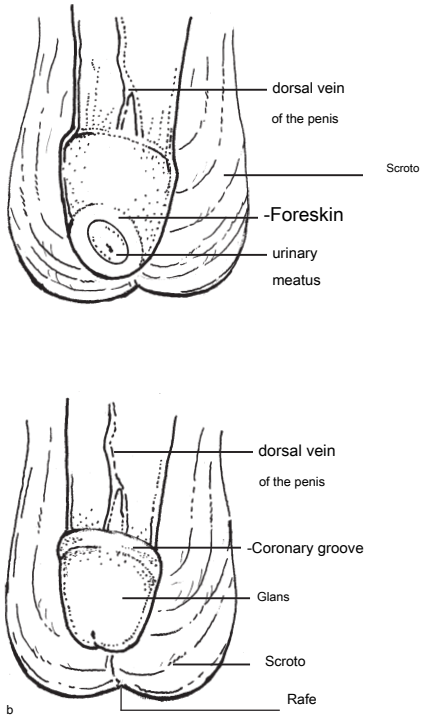


Fig. 19.5 Scrotum and anterior extremity of the penis: a, uncircumcised; b,

ejaculatory duct, which, at the moment of ejaculation, pours it into the urethra.

The vas deferens comes after the tail of the epididymis and extends to the seminal vesicle (see Fig. 19.2).

The seminal vesicles are membranous, extensible and contractile receptacles in which sperm accumulates as it is produced (fig. 19.6).

There are two ejaculatory ducts, one right and one left, and they result from the meeting of the ampulla of the vas deferentes and the corresponding seminal vesicle. They lead the sperm accumulated in these two receptacles to the urethra.

PENIS

The penis is the organ of copulation in men: its function, in the act of intercourse, is to carry sperm to the genital parts of the woman.

The penis is located immediately above the pouches, in front of the symphysis pubis. Located at its beginning in the thickness of the perineum, it is directed obliquely upwards and forwards. Upon reaching the symphysis, it detaches from the deep region, becomes free and is surrounded by a skin cover. It has two portions: a perineal portion and a free portion. The latter, in its flaccid state, appears pendulous, forming with the former the angle of the penis or penile; In an erect state it rises towards the abdomen, then continuing in the direction of the perineal portion.

In the penis, the body and two extremities are considered:

Body. It is cylindrical, somewhat flattened from front to back, with an upper face, a lower face and two lateral edges.

Posterior limb Also called root of the penis;

It is fixed: by the insertion of the corpora cavernosa into the ischiopubic branches: by the suspensory ligament, odd or middle, which starts from the linea alba and the symphysis and ends at the same time on the upper and lower surface of the corpora cavernosa (by fibers that surround it laterally) and by the fibrous ligament of the penis, which goes from the posterior part of the penis to the symphysis and the medial perineal aponeurosis.

Forelimb. It is represented by the glans and its foreskin (see fig. 19.5). The glans is a conoid eminence that offers to our consideration: the apex, directed forward (urinary meatus); the base, cut at the bevel at the expense of its lower face and surpasses the body of the penis by the part we call the crown (the base is correctly limited by a groove, the coronary or balanopreputial groove) and the outer, smooth surface, which presents, in its lower part, a median groove, the frenulum, and its lateral pits.

The foreskin is an integumentary fold arranged in the form of a sleeve around the penis, which is composed of erectile formations and envelopes or covers.
The erectile formations include the two corpora cavernosa and the corpus spongiosum. The envelopes or covers are four, superimposed from the outside to the inside: cutaneous cover, muscular cover, cellulose cover and elastic cover.

Two glands are attached to the male genital tract: the prostate and Cowper's glands.

PROSTATE

The prostate is an organ of glandular nature that develops around the initial portion of the urethra (see Fig. 19.6). It is located in the pelvic cavity, immediately below the bladder. It has the shape of a cone with an upper base and is flattened from front to back. It is gray in color and has a hard consistency. In adults, it measures 28 mm long by 40 mm wide and 25 mm thick.
Its weight is 20-25 g. It should be noted that it is rudimentary in the child, that it grows rapidly at the time of puberty and that it hypertrophies more or less in the elderly.

The adult prostate is in continuous secretory activity, which depends on the degree of androgen stimulation, and produces 0.5-2 mL/day that is eliminated with the urine.

The prostatic secretion that is discharged with ejaculation is a milky fluid that contains citric acid, choline, cephalin and cholesterol, as well as proteins, various enzymes and electrolytes in concentrations similar to those in blood plasma. It also contains calcium, acid phosphatase and fibrinolysin.

GL NODES OF CO PER GL NODES

BULBOURETHRAL

They are very small (3-5 mm) and are located in the urogenital diaphragm, between the fascia and the urethral sphincter.
Its ducts open into the bulbar urethra. They secrete a thin liquid that lubricates the urethra.

PHYSICAL EXAMINATION

The examination of the male genital organs will be discussed in greater depth in another subject of the medical profession (Urology).

General guidelines

Minimize stress

Men are generally less prejudiced than women about issues related to sex and usually do not feel as depersonalized or degraded by this facet of the exam.

Sociocultural factors can influence attitudes, as men are accustomed to nudity in closed rooms and most of them do not feel uncomfortable when examined by male doctors. However, some men may feel tense about the examination, especially if the digital rectal examination is performed in conjunction with the genital examination, much more so if the examination is performed by a woman.

Female examiners may feel some discomfort and tension about the examination of the male genitalia and may transfer their feelings of 'embarrassment' to the examinee. Working on their own feelings and discussing them with their peers, team doctors, and teachers can help build confidence and self-assurance in performing this part of the exam.

menu

Some men may experience an erection during the exam, which can be an embarrassing situation. If this occurs, the examination should continue and the subject should be assured that this is a normal physiological response to genital palpation.

Protection and position

The examiner should wear gloves on both hands during the examination. Gloves offer protection from inadvertent exposure if infection is present. In case of genital infections, gloves should be changed before the rectal examination to avoid cross-contamination.

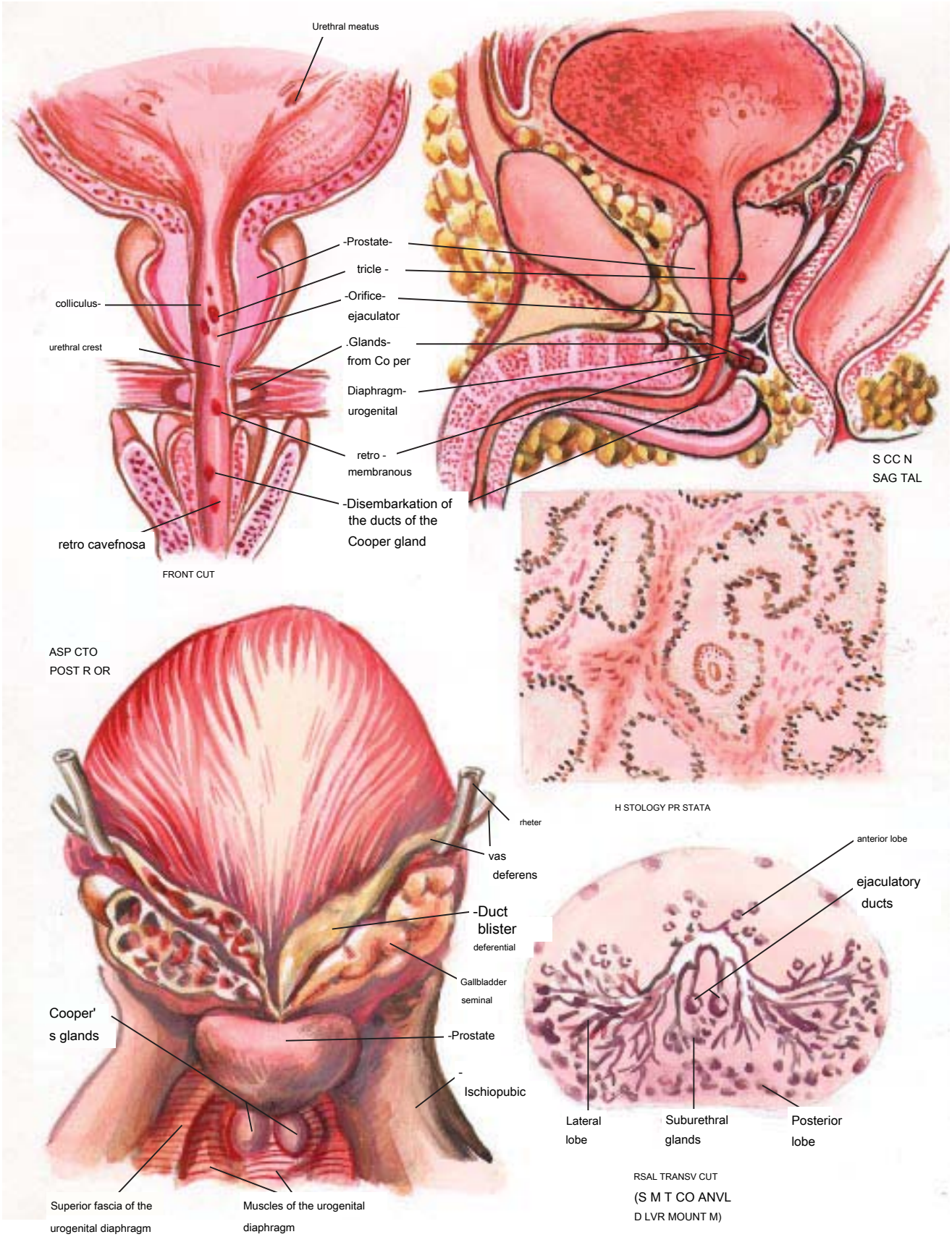


Fig. 19.6 Anatomy of the prostate and seminal vesicles.

For the examination, the man should stand so that the inguinal area can be easily palpated. You should be sitting, facing the person's genitals. The man can wear an examination gown, which is easy to pull up during the exam.

EXPLORATION TECHNIQUES

Examination techniques to evaluate the male genitalia and inguinal region include inspection and palpation.

Inspection

It includes visual examination of the penis, scrotal sacs, inguinal regions, and perineum.

1. Inspect the penis:

a) Ask the person to show his or her genitals by lifting the examination gown and inspect the shape and size of the penis.

b) Inspect the penis and note the integrity of the skin of the glans, foreskin and shaft. If the man is uncircumcised, ask him to pull back the foreskin so you can inspect the underlying area.

The size and shape of the penis varies considerably among adult men, but is not usually the subject of clinical interest. There is much less variation in size when the penis is erect. The glans varies in size and shape and may appear rounded, wide, and even pointed. Help the subject understand normal variations, if he or she expresses interest.

In an uncircumcised child or adult who does not clean the area carefully, smegma may be found, which is a normal discharge that can accumulate on the penis, especially under the foreskin. Smegma results from secretions from Tyson's glands and appears as a thick, white, cheese-like substance. Accumulated smegma can become a culture medium for bacterial growth.

Rule out the presence of ulcerations, which almost always occur in the mucosa of the glans penis, at its base next to the frenulum or in the balanopreputial groove.

c) Inspect the urinary meatus by squeezing the glans between the index fingers and thumb and gently moving them backwards to expose the meatus (Fig. 19.7).

Normally the urinary meatus is free of drainage and secretions. If any secretion is detected, it should be cultured. If the man expresses a history of urethral discharge, but it is not revealed by this maneuver, ask him to squeeze the body of the penis with his fingers.

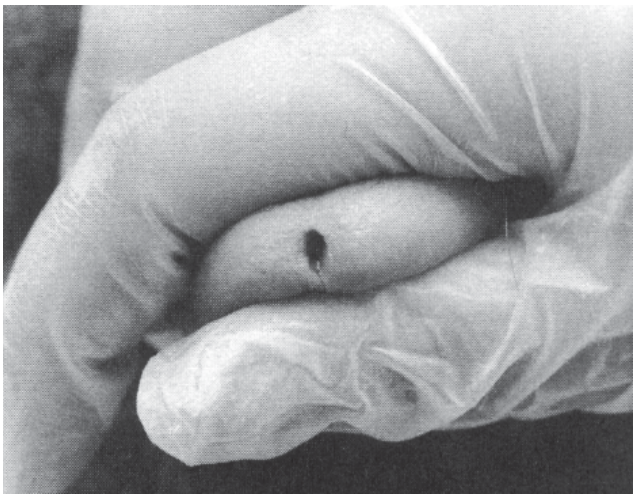


Fig. 19.7 Inspection of the urinary meatus.

penis, from base to tip. If any urethral secretion appears, it should be cultured.

2. Inspect the scrotal bags.

Move the penis to one side, or ask the man to do so, to inspect the scrotal sacs. Then raise the scrotum and inspect its posterior part. Comparatively observe the shape and size of both hemibags and as a whole, and the integrity of the skin.

Normal Findings: The size and shape of the scrotum varies considerably, and may cause fear and discomfort in men who believe the sexual myth that a large scrotum is associated with virility.

Some scrotal sacs hang below the penis, while others are above it.

The left side of the scrotum is usually lower than the right.

The scrotum remains high and appears smaller when the scrotal muscles contract, in response to cold or fear.

The skin of the scrotum has scattered hairs. The skin is thin, and has a wrinkled appearance.

Lesions should be recorded as abnormal findings. If there is an increase in size of one of the two compartments, its origin must be determined later, by palpation.

Palpation

It must be ordered and comparative. It successively includes:

- Palpation of the penis (optional).
- Palpation of the scrotum.
- Palpation of the tunica serosa vaginalis.
- Palpation of the testicle and epididymis.
- Palpation of the vas deferens and the spermatic cord.

- Palpation of the spongy urethra.
- Palpation of the prostate, seminal vesicles and Cowper's glands.
- Palpation of the membranous and prostatic urethra.
- Palpation of the nodes in the inguinal regions.

1. Palpate the penis.

Palpation of the penis is optional in asymptomatic boys or young men. Palpate the shaft of the penis with your index finger and thumb and check for pain or any mass.

No masses should be palpated along the shaft of the penis and it is normally not painful on palpation, nor when gently squeezed to access the meatus.

2. Palpate the scrotal sacs.

Normally there is no difficulty in reaching the organs (testicles, epididymis, spermatic cord) located inside the sacs:

a) Palpate one scrotal compartment at a time, pinching the scrotum between your thumb and index finger. Rule out the existence of edema, emphysema, varicocele and tumors.

In the case of scrotal edema, the skin offers a pasty resistance and leaves a mark (pit or godet) to pressure.

In emphysema (pneumoscrotum) a slight crepitation is palpable (as if small dry and friable vesicles were crushed). The varicocele awakens a special sensation compared to that caused by a bunch of worms or chicken guts.

Benign tumors (for example, sebaceous cysts), malignant ones, syphilitic chancre, etc., stand out well for their hardness.

b) Palpate the tunica serosa vaginalis, looking for the sign of impingement of the vaginal serosa.

Try to grasp the anterior part of the testicle between the thumb and index finger, which, when the vaginal cavity is not distended by an effusion, flees in front of the fingers, which only manage to grasp the wall of the sacs, including the parietal sheet of the vaginal serosa, which forms a fold, and which, if the pressure is increased, detaches while at the same time a sensation of jumping is perceived.

The sign of impingement of the vaginal serosa contributes to establishing the differential diagnosis between pachyvaginalitis or hematocele of the vaginal serosa and testicular tumors.

c) Squeeze gently to detect the testicle, remembering that this maneuver may be slightly painful.

Normal findings: two ovoid-shaped testicles should be palpated through the skin of the scrotum,

Fig. 19.8 Palpation of the epididymis.

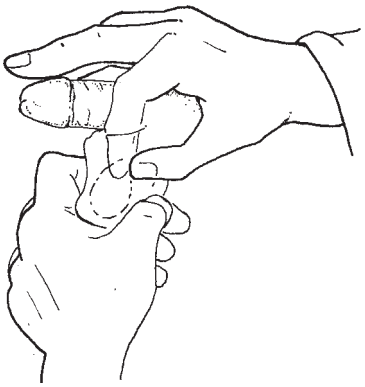
smooth surface with a homogeneous, firm and elastic consistency, which resembles that of the eyeball in the living animal. The testicles must move freely. After puberty, percussion or compression of the gland is painful and should be avoided.

d) Palpate the epididymis by pinching the back of the scrotum between the thumb and index finger and feel it firm, with a comma-like structure (Fig. 19.8).

The epididymis attaches in the shape of a comma and circumvents the testicle. Its tail is continuous with the vas deferens, which, together with the neurovascular elements, constitutes the spermatic cord.

The epididymis is less consistent (meager thickness of its fibrous tunic) and is well palpated with the Chevassu technique: while the testicle is fixed with the left hand, which pulls it downwards, the right index finger is hooded in the skin of the lateral aspect of the penis in front of the scrotum, trying to pinch the head of the epididymis between it and the thumb of the same hand (fig. 19.9).

Fig. 19.9 Palpation of the epididymis (Chevassu technique).



e) Finally palpate the vas deferens and the spermatic cord comparatively, moving your thumb and index fingers from the epididymis to the duct, in an anterior direction. Palpel them up to the inguinal ring.

The vas deferens, which is well isolated from the other elements of the spermatic cord, is cylindrical, hard, smooth and with a diameter between 2 and 3 mm.

The elements of the spermatic cord should feel like a freely movable cord.

The epididymis and vas deferens should be palpated without masses and should not be painful.

3. Palpate the spongy urethra.

Raise the penis, bringing it closer to the abdominal wall, to directly reach the spongy portion of the urethra, or palpate it through the pouches or anterior perineum, to rule out the presence of soft or hard infiltration areas.

4. Palpate the prostate, seminal vesicles, and Cowper's glands.

The prostate examination was already explained along with the digital rectal examination in Chapter 16.

Remember that in healthy subjects it has an oval shape (with a longitudinal groove on its back), a uniform and elastic consistency under tension, very precise limits, little mobility, and it is painless. Its length is estimated at about 3-4 cm. Its expression gives rise to white drops at the level of the urethral meatus.

5. Palpate the membranous and prostatic urethra.

The membranous and prostatic portions (between the neck of the urethral bulb and the bladder) are palpated by rectal examination, to rule out the presence of soft or hard infiltration areas.

6. Palpate the nodes in the inguinal regions.

Palpation of the inguinal lymph nodes was already addressed in Chapter 17, corresponding to the hemolymphopoietic system. We will only add some points.

Both the superficial and deep nodes collect lymphatics from the skin of the scrotum and those from the penis. There is a deep one, known as Cloquet's ganglion, whose inflammation has been able, in certain cases, to cause accidents that are entirely reminiscent of hernia strangulation.

The lymphatic drainage of the testicles is carried out towards the abdominal cavity, so alterations in these gonads rarely have expression in the inguinal lymphatics.

7. Inspect and palpate for hernias.

The examination of the inguinal regions looking for hernias has no direct relationship with the genital examination, but these regions are considered by some as "no man's land", which can cause diagnostic problems.

tics, which may even endanger a person's life, if it is not taken into account during inspection and palpation, in the regional examination of the abdomen. What happens is that the examination of the inguinal regions is never complete if the presence of a hernia is not ruled out, which is usually done together with the examination of the genitals; and if their exploration is deferred, valuable data from this region may escape.

Although part of the examination of these regions was already mentioned, in the peripheral arterial system, for the exploration of the iliacofemoral vessels, and in the examination of the lymphatic system, for the exploration of the nodes at that level, we will now insist on the examination of the inguinal regions in men, fundamentally, for the search for inguinal and inguinoscrotal hernias, usually these alterations in asymptomatic individuals:

a) Inspect the inguinal area for any bulges. Ask the man to bear down and cough so that any protrusion can be detected.

Normally no bulges should be seen over the inguinal and femoral canal, at rest or during coughing, sneezing or pushing.

b) Palpate the area over the femoral canal, with and without the man pushing.

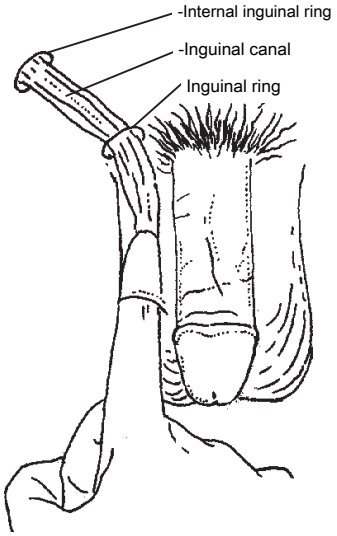
c) Palpate the inguinal canal (fig. 19.10):

To palpate the right inguinal canal, approach the man on the right side.

Palpate the inguinal canal by invaginating the loose skin of the scrotum with your right index finger, from the scrotal fornix.

Follow the spermatic cord with your finger toward the external inguinal ring, a triangular, slot-like opening.

Fig. 19.10 Palpation of the inguinal canal.



If the inguinal ring is large enough,
Continue advancing your finger through the inguinal canal.

Ask the man to cough or bear down.
Repeat the procedure on the opposite side.
Palpable masses in the inguinal region may represent hernias.

Inguinal hernias

Indirect inguinal hernia. It is a herniation through the inguinal canal. It may be felt with the tip of the finger as a lump in the canal or it may extend through the canal into the scrotum. Indirect inguinal hernias can be detected by inserting your index finger into the inguinal canal.

Direct inguinal hernia. It does not travel through the inguinal canal; on the contrary, the inguinal sac protrudes anteriorly through the abdominal wall. During palpation of the inguinal canal, the hernia moves the examining finger forward. Alternatively, a direct inguinal hernia may feel like a bulge between the thumb and index finger when the skin around the external canal is palpated as the person pushes.

Direct hernias rarely descend into the scrotum.

Femoral hernia. Femoral hernia can be detected below the inguinal ligament and medial to the femoral pulse, as a visible or palpable bulge. Femoral hernias are more common in women.

TESTICULAR SELF-EXAMINATION

Students in the clinical area must know from the beginning how a testicular examination is performed to teach it to the male population at the appropriate time. The physical examination of the reproductive system is an opportune time to promote health, since testicular cancer is the main cause of death from cancer among male adolescents and young adults; However, if treated early, the chance of survival is almost 100%.

The American Cancer Society recommends testicular self-examination, at least monthly, for early detection of testicular cancer.

HIGH RISK GROUPS

The risk of developing testicular cancer is highest in men 15-34 years of age. Boys and men of any age with an undescended or partially descended testicle have a higher risk of testicular cancer.

EXPLORATION TECHNIQUE

The testicular self-examination lasts 3-4 minutes and should preferably be performed after a warm bath or shower, when the skin of the scrotum is relaxed and easy to manipulate.

1. Examine the left testicle with the left hand, while lifting the penis with the right hand. Palpate the entire testicle, taken between the thumb and index finger, rolling the skin that covers it with them in a horizontal plane (fig. 19.11). Look for the presence of nodules or other abnormalities.

2. Repeat the procedure moving the fingers in a vertical plane (fig. 19.12).
3. Examine the right testicle with the right hand, using the same procedures.

Fig. 19.11 Palpating in a horizontal plane.

Fig. 19.12 Palpating in a vertical plane.

It is normal to find one testicle larger than the other.
If any hard, nodular area is found, it should be reported to the health professional.

An established testicular tumor is palpated as an irregular, painless, fixed mass. Feelings of heaviness or discomfort have been reported. Enlargement of the lymph nodes in the region is rarely seen, because lymphatic drainage from the scrotum and testicles extends into the abdominal cavity.

20

ANATOMY AND PHYSIOLOGY OF THE FEMALE GENITAL ORGANS

For a better understanding of the alterations produced in this system by the different processes or conditions, it is necessary to carry out an account of the fundamental aspects of its anatomy and physiology.

ANATOMICAL SUMMARY

Ovary fig

It is a paired and symmetrical organ, located in the minor pelvis in the so-called ovarian fossa, which borders behind the internal iliac vessels, in front with the infundibulopelvic ligament and above with the innominate line of the coxalis. It lies behind and to one side of the uterus, attached to it by the uteroovarian ligament and laterally attached to the broad ligament by the mesovarium, which continues with the infundibulopelvic ligament when reaching the pelvic wall. Despite these means of support or fixation, the ovary is a very mobile organ that oscillates in its place and can even change location.

The ovary has a flattened ovoid shape, with a greater diameter of 3-5 cm, a transverse diameter of 2-3 cm and a thickness of 1-3 cm. These diameters are smaller in girls, increase during puberty and towards adulthood, and decrease during menopause.

Trumpet see fig

It is a paired organ that is found on both sides of the uterus, with which it communicates and continues at the level of the uterine horn that extends laterally to the ovary, near which it ends and to which it is attached by the tube-ovarian ligament. It has a length of 9-12 cm and is shaped like a tuba or trumpet. It is divided into four portions: interstitial (through the uterine wall), isthmic (at the exit of the uterus), ampullary (intermediate, the most extensive) and fimbric or pavilion (tongues at the free abdominal end). The diameter of the tubal lumen is 1 mm in the interstitial portion, slightly more in the isthmic portion, and 4 mm in the ampullary portion.

The proboscis is located in the middle fin of the broad ligament. It should be kept in mind that during pregnancy, the tubes and ovaries rise with the fundus of the uterus in the abdominal cavity.

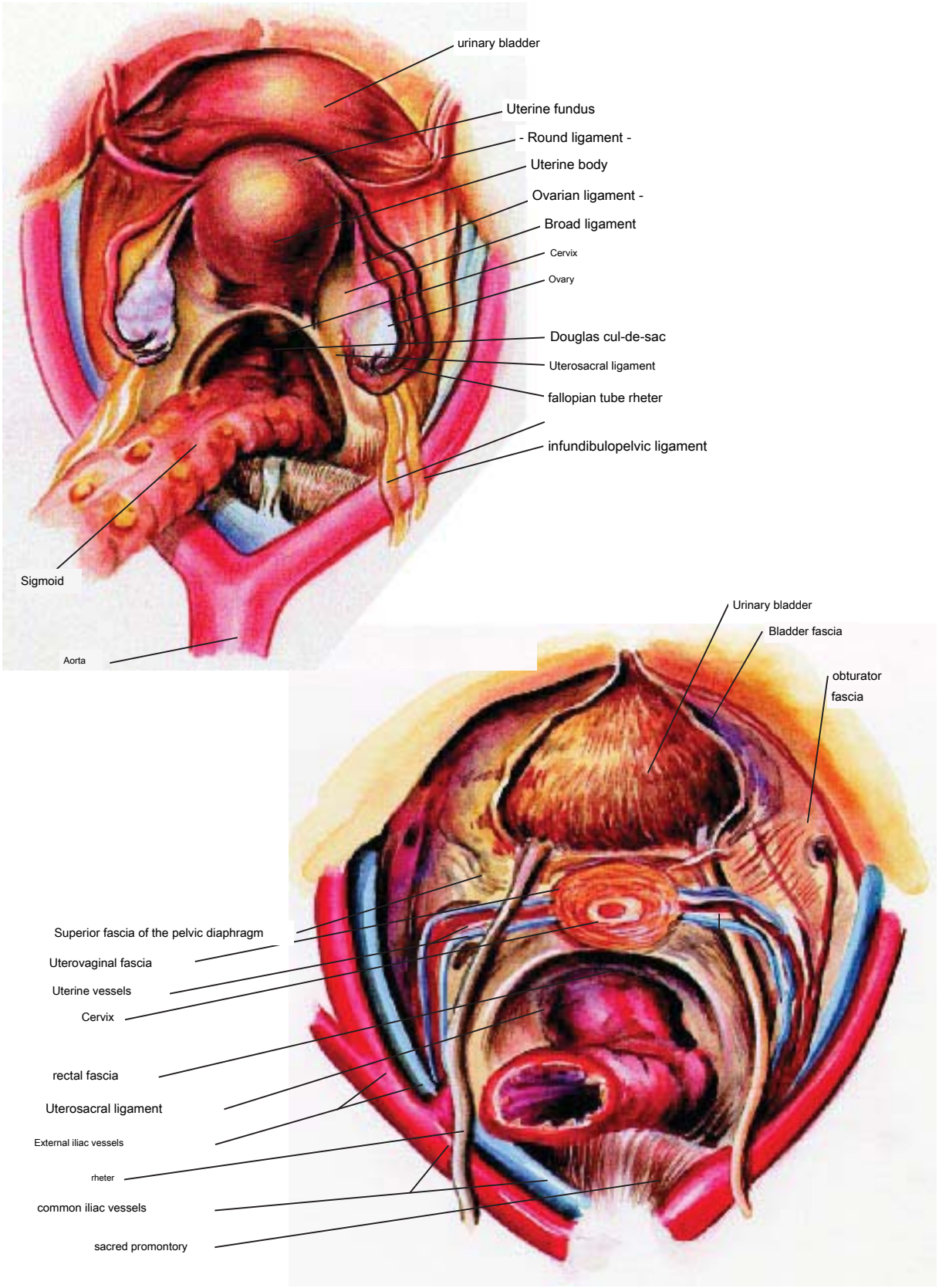


Fig. 20.1 Female internal genitalia and supporting ligaments.

tero see fig

It is an odd organ, located in the center of the bony pelvis and fixed to it by the lateral or Mackenrodt ligaments as fundamental elements, and secondarily by the uterosacral ligaments, behind, and to a lesser extent by fibers that join it to the bladder and the pubis in front (uterovesicopubic ligament).

The upper portion or body, 4-5 cm in longitudinal diameter, is shaped like an inverted pear, flattened from front to back, and has an isosceles triangular cavity with an upper base and whose angles coincide with the beginning of the tubes; This cavity is covered by the endometrium and has a capacity of 4-6 mL, which can increase in multiparous women.

The body is flattened from front to back and triangular in shape; It has two faces and three edges. The anterior surface, slightly convex, is covered by the peritoneum in its entirety and corresponds to the bladder from which it is separated by the vesicouterine cul-de-sac (frequent presence of intestinal loops in the cul-de-sac). The posterior surface, more convex than the anterior, has a blunt ridge in its middle part that corresponds to the rectum, from which it is separated by the rectovaginal fornix. The lateral edges, oblique from top to bottom and from outside to inside, correspond to the broad ligament, the uterine artery and the venous plexus that accompanies it. The lower extremity merges with the neck at the level of the isthmus and the upper extremity or fundus, concave in the fetus, rectilinear in the nulliparous, convex in the multiparous, corresponds to the intestinal loops.

The lower portion or neck is 3 cm long with a duct 3-5 mm in diameter, an upper limit (internal cervical orifice) that communicates with the uterine cavity and a lower limit (external orifice) that communicates with the vagina.

The transition zone between the neck and the body, called the isthmus, is located somewhat above the internal cervical opening and is of great importance in pregnancy and childbirth, since it will give rise to the so-called lower segment.

The vaginal dome or upper limit of the vagina is fixed to the neck, delimiting a supravaginal portion and another intravaginal portion or tench snout, which has the shape of a cone whose vertex, directed downwards, is truncated and rounded. At this vertex, a hole 4-6 mm in diameter is seen, the external hole of the neck. This portion is visible on speculum or leaflet examination.

Vagina

It is a tubular organ in a state of distension and flattened from front to back under normal conditions;
presents a virtual cavity. The longer posterior wall can measure 8-10 cm and the anterior wall 6-8 cm. His Distended cavity during intercourse or by a speculum is about 3-4 cm in diameter and during childbirth it can reach 10-12 cm.

The lower extremity of the vagina is continuous with the vulva in the so-called vaginal opening or introitus and the upper extremity is a circular opening that embraces the cervix, at the junction of its lower third with its upper two thirds, and adheres to it intimately. Thanks to this adhesion, there is a circular canal around the entire contour of the tench snout, between it and the vagina, which is called the vaginal vault or vaginal cul-de-sac.

This pericervical canal is divided topographically into four parts: a shallow anterior part or fornix, which corresponds to the bottom of the bladder and the vesicovaginal arteries; a posterior part or posterior fornix, which is 10-25 mm deep and corresponds to the fornix of Douglas and the rectum; and two lateral parts or lateral fornices, run through the ureter, the vaginal plexus and three or four lymphatic ducts. (It should be noted that a space of 10-15 mm ordinarily separates the lateral fornix from the uterine artery.)

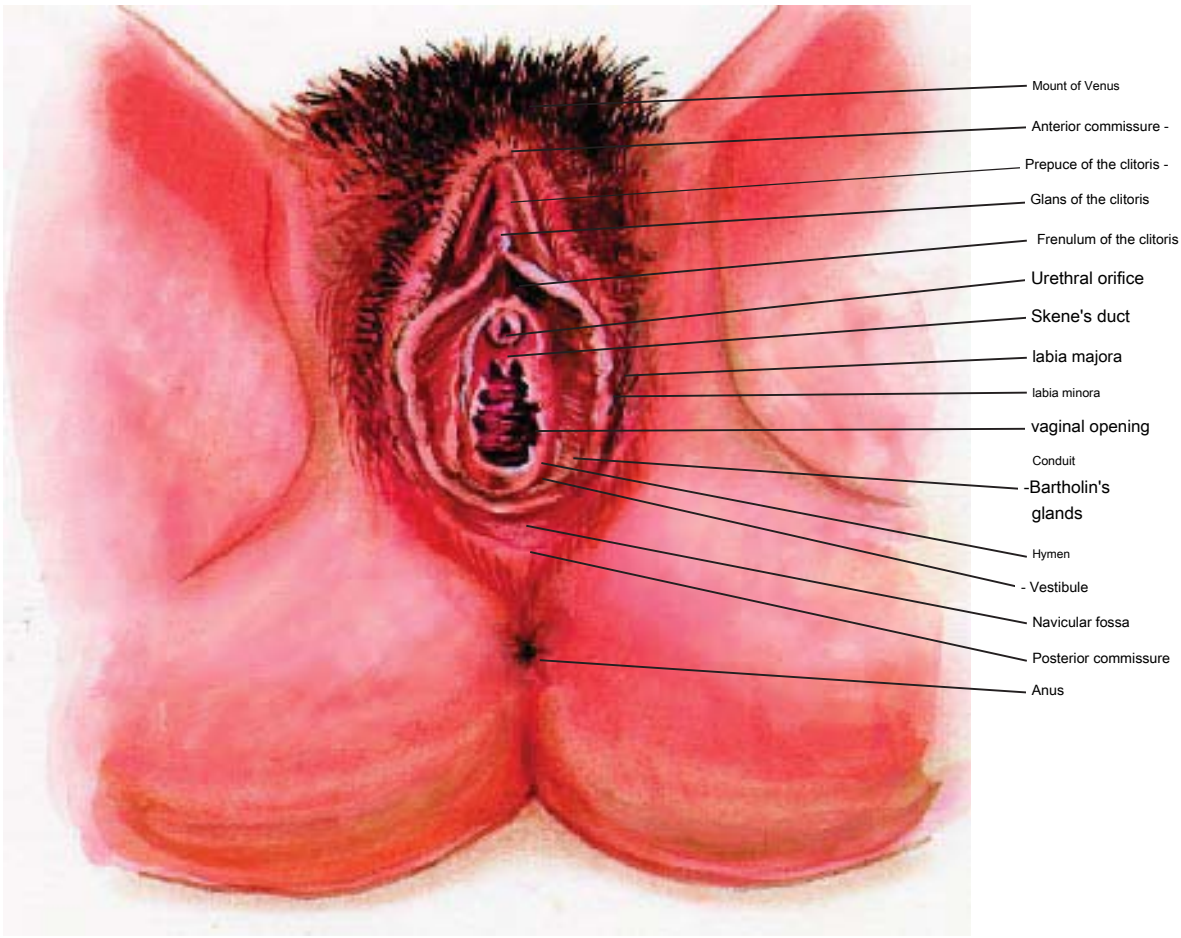
The tunic mucosa, approximately 1 mm thick, grayish or slightly pink (red during menstruation and pregnancy), very resistant, very extensible and very elastic, is composed of a chorion and a stratified squamous epithelium. It is devoid of glands.

Vulva

It is a wide opening found in the perineal region, between the mons pubis in front and the anus behind, and is covered with hair around it (fig. 20.2).

On both sides it has two folds or rings of skin with a large amount of cellulose fatty tissue, the labia majora that, meeting together in the midline, form the commissures of the vulva: anterior conmissure, rounded in the shape of an arc, which is continuous with the mons of Venus, and posterior commissure or fork, thin and very apparent, which is separated from the anus by the perineum or perineum (in front of it the navicular fossa).

Inside the labia majora there are two minor folds that do not have fat inside, the labia minora or nymphs, which continue upwards, closing an angle that constitutes the hood of the clitoris, or prepuce that covers this erectile organ of the woman, with a cavernous structure similar to the penis. Behind the clitoral hood the external orifice of the urethra opens, which is masked by the aforementioned structures and the labia minora. Behind the external opening of the urethra and delimited by the labia minora we find the vaginal introitus, which in women



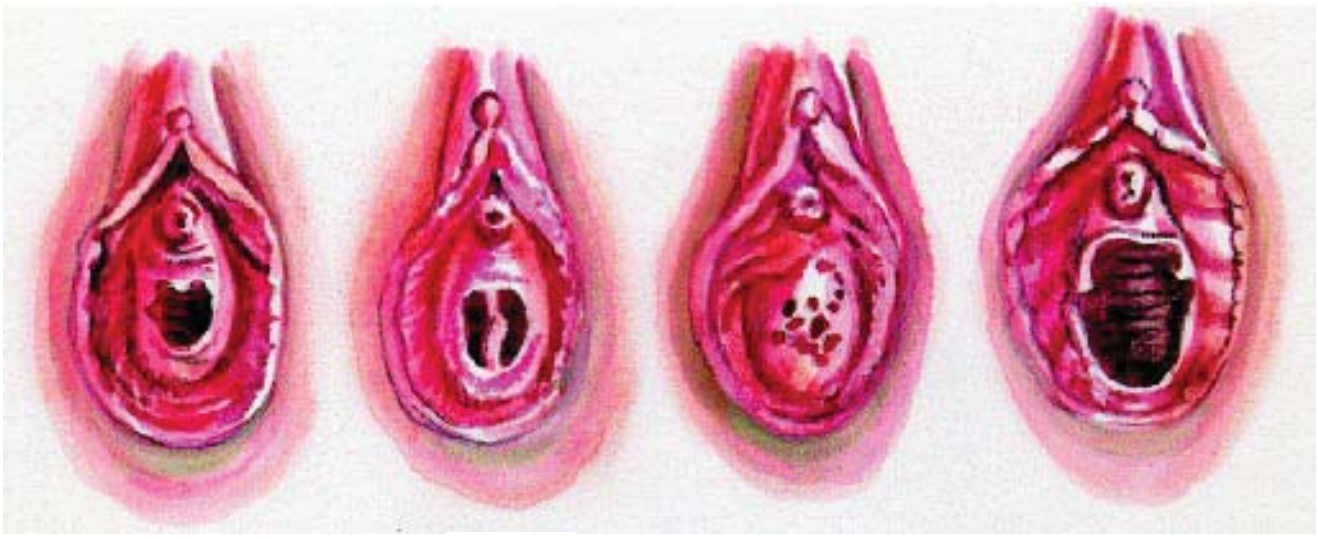


Fig. 20.2 Female external genitalia.

It may be occupied by an incomplete membrane, the hymen, which partially disappears with sexual relations and sometimes almost completely with childbirth, leaving only small remains called myrtiform caruncles.

The labia minora end imperceptibly behind on the inner surface of the corresponding labia majora.

The area framed by the labia minora and the clitoris and its hood is also called the vestibule of the vulva and into it the vestibular or Bartholin glands open on both sides, which lubricate the genitals during copulation, and on both sides of the urethra the periurethral glands open, which are the homologues of the prostate in the female.

Pelvic floor or perineum

The woman's perineum or perineum has the same shape, the same limits and also approximately the same fundamental constitution as that of the man.

It is made up of nine muscles and three aponeurosis that are arranged in three planes: superficial, middle and deep.

Superficial plane (fig 20 3)

It is made up of the following muscles:

Bulbocavernosus. It surrounds the vulva, going from the corpora cavernosa of the clitoris to the tendinous raphe of the perineum.

Ischiocavernous. It goes from the corpora cavernosa of the clitoris to the ischial tuberosity.

Superficial transverse perineum. From the ischial tuberosity to the tendon raphe of the perineum.

These three muscles form a triangle on each side of the vulva, leaving a fourth muscle, the striated sphincter of the anus, which runs from the tendonous raphe of the perineal region to the back, surrounding the anus and fixing on the coccyx.

Medium plane (fig 20 4)

Consisting of the deep transverse perineum (with a similar arrangement to the superficial one), which extends forward in the form of a fibrous diaphragm, which is inserted into the ischiopubic branches above the ischiocavernosus, called the urogenital fascia or urogenital diaphragm, which gives an envelope to the urethra forming its striated sphincter.

deep plane

Formed by the levator ani muscle or main pelvic diaphragm. It runs from the sacrococcygeal raphe and the coccyx, fanning forward until it inserts into the ischial tuberosity (ischiococcygeal bundle); a tendinous arch that is formed from the fascia of the obturator muscle (ileococcygeal bundle) and, finally, in the pubis (pubococcygeal bundle) whose innermost fascicles are sometimes puborectalis. This muscle has the shape of a funnel when viewed from above and in its anterior part in the midline it leaves an opening, the urogenital hiatus, through which the vagina and urethra pass, which will be occluded by the urogenital diaphragm (fig. 20. 5).

PHYSIOLOGICAL SUMMARY

The cerebral cortex, the hypothalamus and the adenohypophysis are involved in the neuroendocrine regulation of the sexual cycle in women.

At the level of the hypothalamus, release factors or release hormones have been demonstrated that, acting on the pituitary gland, regulate the production of gonadotropins: follicle-stimulating (FSH) and luteinizing (LH).

Starting from the beginning of menstruation and through the action of the aforementioned release factors, the pituitary gland stimulates the ovary through follicle-stimulating hormone (FSH), inciting the growth and development of several follicles and their estrogen production, which will progressively increase until the high level of estrogen in the blood, through feedback, causes the hypothalamus to inhibit the production of FSH and stimulate the production of LH, which together participate in the mechanism of follicular rupture and ovulation (day 14 of the cycle).

The rupture is influenced by the decrease in blood flow in the most superficial area of the follicle together with the vasoconstriction produced by prostaglandins to form the stigma, through which ovulation occurs. The luteinizing hormone stimulates the transformation of the follicle (before it ruptures) into the corpus luteum, which produces progesterone and estrogens that inhibit, through the centers of the posterior hypothalamus, FSH and LH, so the luteinizing action declines and the function of the corpus luteum decreases, causing an estrogen-progesterone hormonal imbalance, which triggers the complex mechanism of menstruation and stimulates the secretion of FSH that restarts the growth of new follicles for the next cycle.

Endometrial cycle and menstruation fig

From the first to the fourth or fifth day of the menstrual cycle, shedding of the functional layer of the endometrium occurs; This is followed by the healing of the endometrial surface due to the estrogenic stimulus and at the expense of the proliferation or growth of the endometrial stroma (glands and vessels) in the proportional relationship 1:2:3 respectively. The glands appear at this stage as straight tubes.

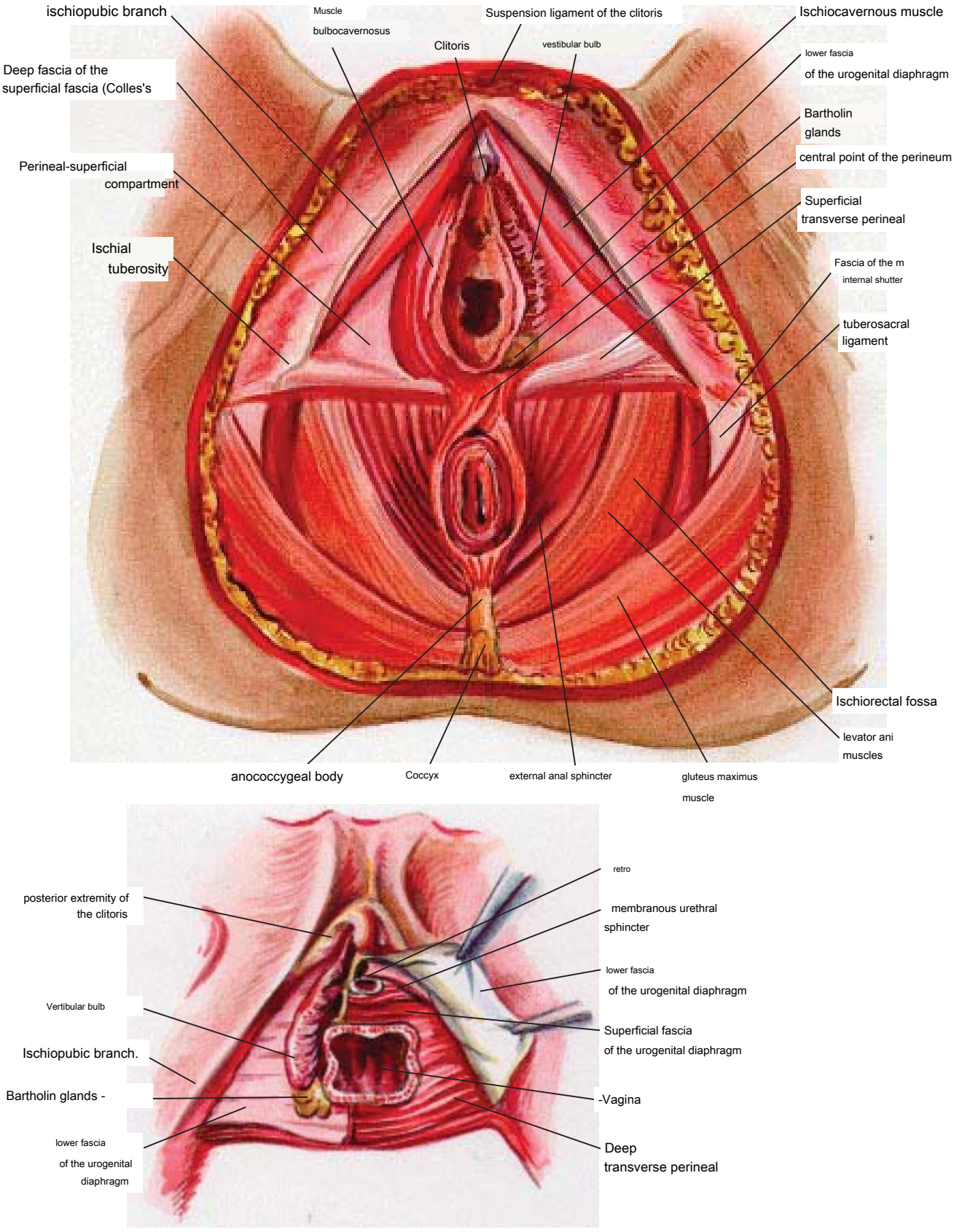
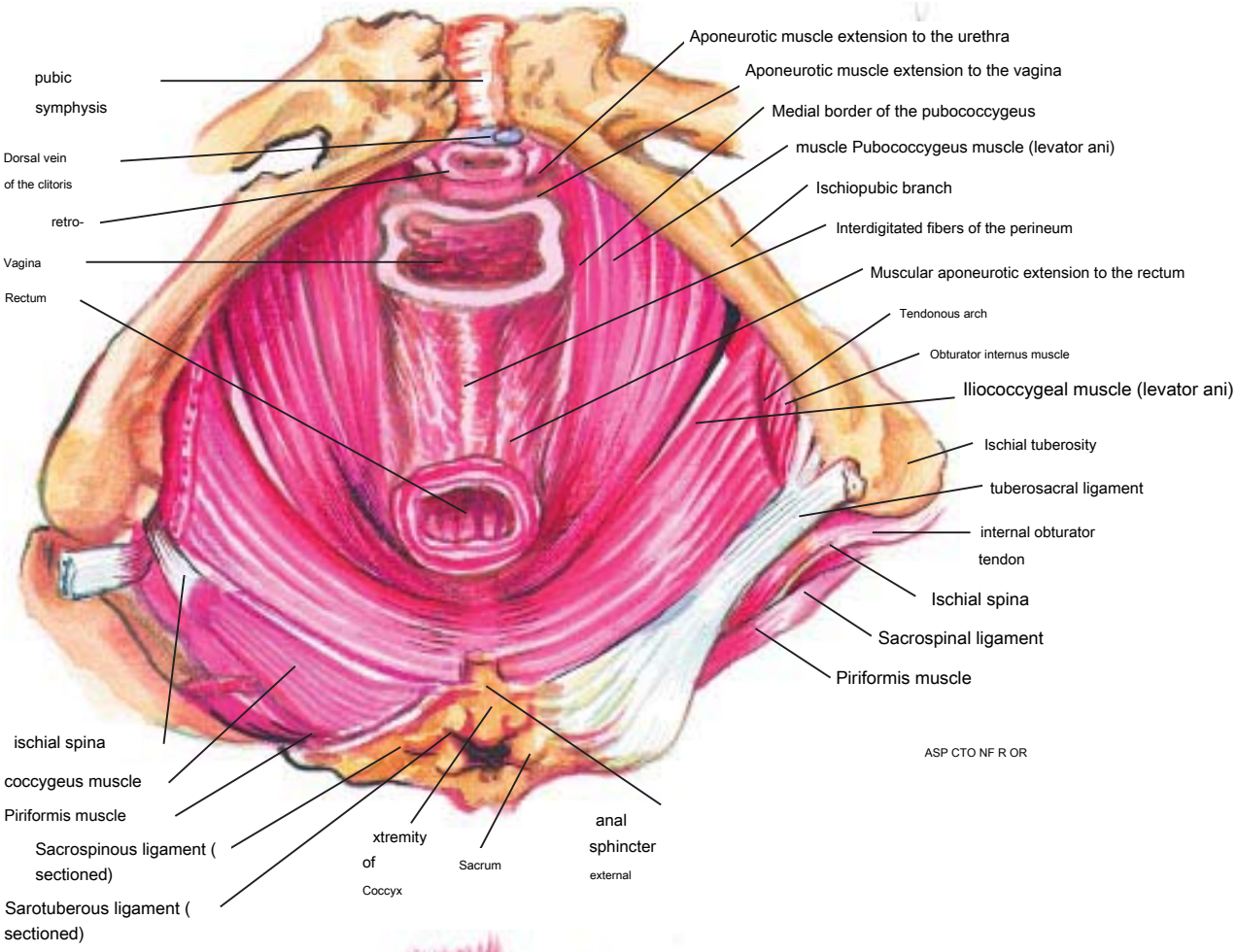


Fig. 20.3 Superficial plane of the perineum.



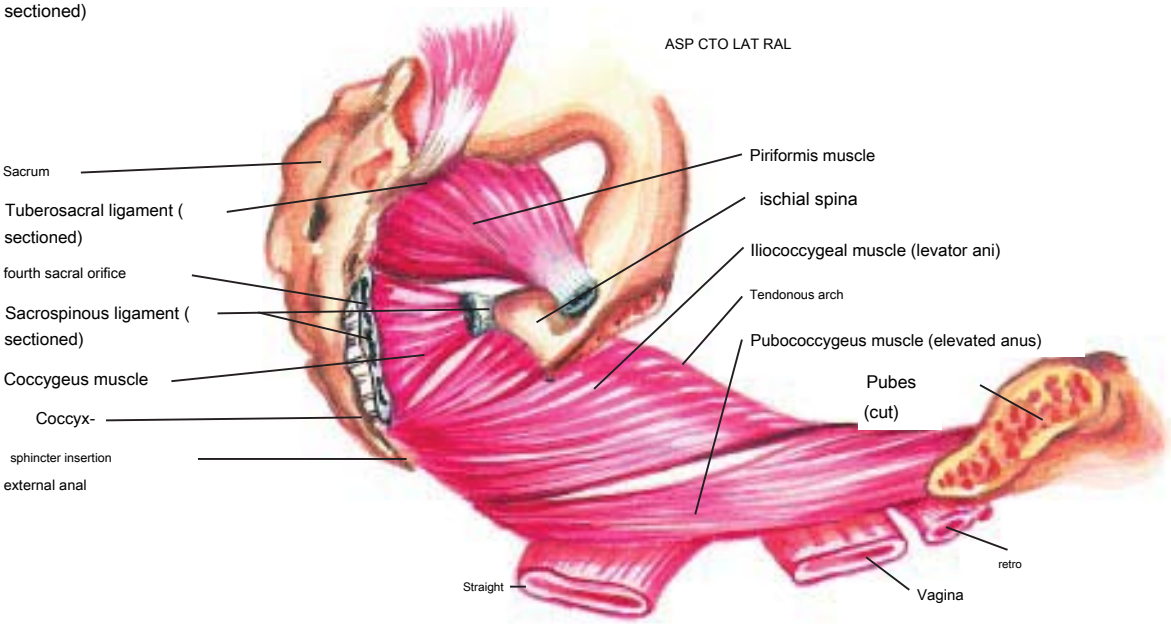


Fig. 20.4 Median plane of the perineum.

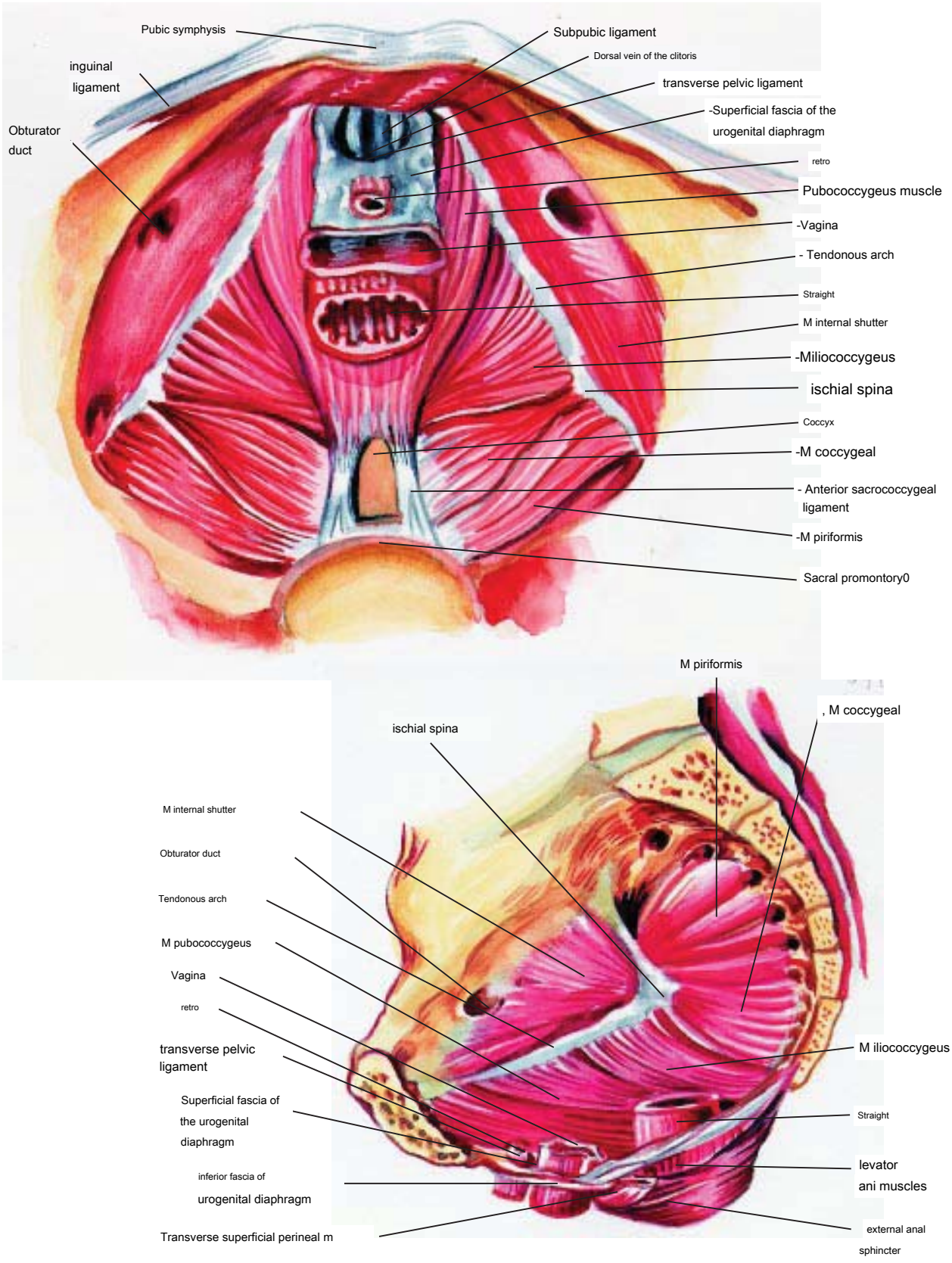
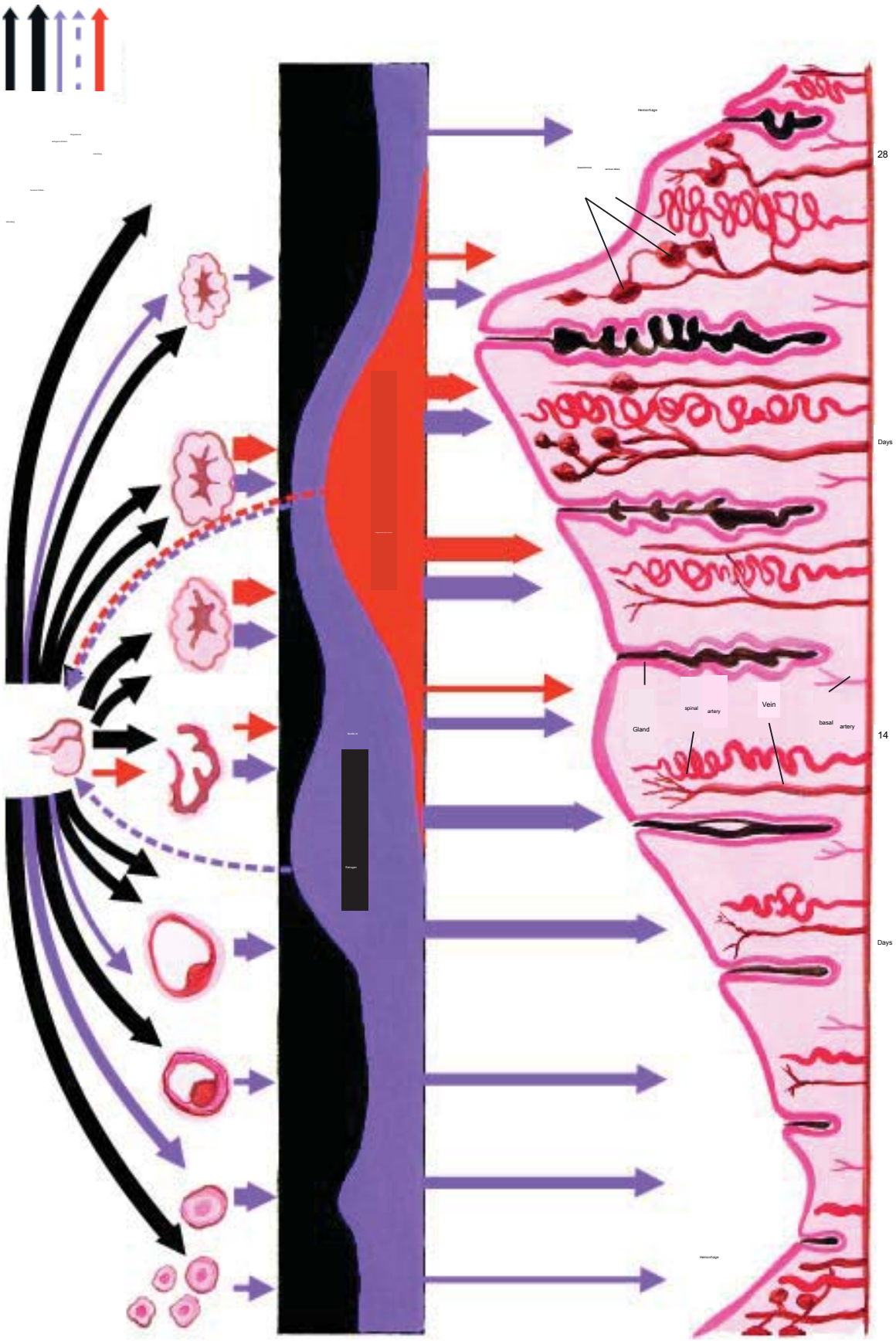


Fig. 20.5 Deep plane of the perineum.


28
Days
14
Days
spinal
artery
Vein
basal
artery
Gland

After ovulation and the formation of the corpus luteum, secretory changes occur in the endometrium due to the action of progesterone. In this secretory phase, the glands become tortuous, vacuolization of the glandular cells appears, epithelial pseudostratification, the nuclei are located basally, the stroma becomes edematous, becoming more lax, and the arteries adopt a spiral shape. In this phase, prostaglandin is synthesized by the action of progesterone. If implantation of the egg does not occur (which, due to the stimulation of chorionic gonadotropin, would maintain the corpus luteum), hormonal decline will occur at the end of this phase. The endometrial cells shrink, endometrial thinning and cell autolysis progressively occur with the release of prostaglandins that cause the contraction of the arterioles with a decrease in their caliber, which produces foci of ischemia.

Loss of stromal fluid further flattens or shrinks the endometrium and aggravates blood stasis.

Subsequently, vasoconstriction of the spiral arteries occurs at their origin and in the myometrium, which, when it temporarily ceases, gives rise to menstrual bleeding, which together with endometrial peeling constitutes menstruation.

Neck

The changes that occur in the endocervix are parallel and are manifested by changes in the characteristics of the cervical mucus, which as the proliferative phase progresses is more abundant, fluid and stringy and if we spread it on a sheet, letting it dry, it crystallizes in the form of fern leaves; All this occurs at the expense of the influence of estrogens. In the second phase of the cycle, the filance, fluidity, crystallization and penetrability of cervical mucus rapidly decrease, as progesterone inhibits the action of estrogens on it.

Vagina

The estrogenic stimulus activates the growth, maturation and shedding of the vaginal and exo-cervical epithelium, so during the proliferative phase epithelial shedding, vaginal acidity, the number of mature cells in the superficial layer and the thickness of the epithelium progressively increase; This is evidenced by the cyclic study of the vaginal contents and can be represented by the so-called cornification curves or the percentage of karyopyknotic superficial cells, which also increases the acidophilic index.

The cells appear more dispersed and isolated, and the number of leukocytes is lower, the vaginal contents are " cleaner." In the second phase of the cycle, the detached cells unite more, constituting true groups.

and they adopt special folded or boat-shaped shapes, desquamation decreases somewhat, leukocytes increase and intermediate type cells predominate, the spread becomes more "dirty". The karyopynotic and acidophilic indices decrease.

PHYSICAL EXAMINATION

GENERAL INSTRUCTIONS

The female genitalia and pelvic structures are examined by simple inspection, by inspection using the vaginal speculum, and by palpation.

At the beginning of the examination, you should put gloves on both hands. Subsequently, for bimanual palpation, the glove can be removed from the hand used to palpate the abdomen.

The examination begins with inspection of the external genitalia and continues with palpation of the vaginal orifice, vaginal speculum inspection of the vagina and cervix, bimanual palpation to evaluate the internal pelvic structures, and, in some cases, rectovaginal palpation.

1. Minimize tension.

Women often have negative feelings about genital and pelvic exams, and may even avoid recommended health screening practices, such as the Papanicolau (Pap) test for cervical cancer (known to us as cytology testing or vaginal cytology), because they fear the exam or, paradoxically, a positive result.

Some women feel tense or uncomfortable about genital examination, because they think that their genitals are dirty or deformed or they are afraid that their genitals will emit unpleasant odors or secretions; or they may see the gynecological position as something with sexual implications, or that the position can produce feelings of helplessness and vulnerability. On the other hand, many women reject the pelvic exam because they feel a loss of control over what happens during the exam (Domar, 1986).

The loss of eye contact between the examiner and the examinee can inhibit communication, causing feelings of depersonalization. The fact that the woman is usually fearful and tense during the examination can be prevented by showing her and/or explaining everything that is being done. Feelings of fear and anxiety may be due to a lack of information and knowledge about the procedure.

You must give security to the woman, offering her confidence and promoting a warm and friendly atmosphere, which allows her fears and judgments to be removed.

Because women generally view genital examination as something unpleasant to endure, you should make every effort to reduce this tension by providing adequate explanations and being as sensitive as possible. Explain the exam, its purpose, the basic steps of the procedure and any discomfort that may arise, before the woman assumes the gynecological position, so that she has the opportunity to anticipate what will happen and. therefore, maintaining a sense of personal control of the situation. You should also give him basic instructions for relaxing the pelvic muscles, which will help save time for when relaxation techniques need to be performed during the exam.

One way to help understand the exam is to give the woman a hand mirror to hold so she can see her genitals as you explain their anatomy, physiology, and procedure. If she prefers not to see her genitals in the mirror, you should respect her decision.

Additionally, if she wishes to have someone present during the exam, every effort should be made to try to accommodate this request. It is advisable for male examiners to take the exam accompanied by a woman. In that case, any conversation between the examiner and the companion must include the examinee. Joking with the examinee or anyone present during the exam causes a feeling of degradation or discomfort in the woman.

2. Preparation and position.

Before the pelvic exam, tell the patient to empty her bladder.

Trying not to injure the woman's modesty, proceed to place her in a gynecological or lithotomy position. Help him into position, with the help of the nurse, if available. Place your feet in the stirrups to maintain leg position.

Although the gynecological position is not comfortable, it is the most effective for the pelvic examination. In some circumstances, stirrups are not used, such as when there is limited joint movement or during labor. Instead, it can be replaced by a modified gynecological position, in bed.

When using the stirrups, the buttocks should be on the edge of the table, with the knees raised and directed to the sides and the heels in the stirrups.

The woman can wear stockings or shoes to keep warm and prevent additional muscle tension, which can increase discomfort. Tension of the abdominal muscles can also be reduced by placing a small pillow under the woman's head and instructing her to place her arms at the sides. sides or cross them over the chest. You should not lie with your arms above your head.

You should sit facing the genitals while inspecting them and when examining them with the speculum. You should stop during the last half of the exam to perform bimanual palpation.

You should cover the woman's upper body with the examination gown, a sheet, or your own clothing if other areas of the body are not being examined.

The woman should be covered with a small part over her knees and moving the center of the cloth down enough to allow eye contact between both. You should observe his facial expression, looking for signs of pain or anxiety. If the woman wishes to observe the exam, it is not necessary to cover her.

Good lighting is necessary, especially when inspecting the cervix. A gooseneck lamp, next to the examiner or at the end of the examination table, is usually sufficient.

3. Hygiene techniques.

A water-soluble lubricant should be applied to gloved fingers to facilitate vaginal penetration only during the bimanual examination. If the lubricant is obtained from a large tube that is used repeatedly, the tube should be squeezed in a way that allows the lubricant to fall onto the gloved fingers. These should never stick to the opening of the tube, which could contaminate its contents.

The vagina should never be examined after the rectum, unless you wash your hands again and change gloves. If a vaginal infection is suspected or present, gloves should be changed between vaginal and rectal examinations to prevent inadvertent spread of the infection to the rectum.

After the exam, clean any excess lubricant from the genitals with cloth or gauze, using a firm sweep from the pubis toward the rectum. The woman may wish to clean the area herself.

EU APPROACH TO EXAMINATION AND WRITTEN RECORD

-External genitalia: color and pigmentation, shape and symmetry, secretions and lesions.

- Vaginal structures: integrity of the skin, position and shape of the neck, color, characteristics of the cervical orifice, lesions or secretions of the neck, vaginal muscle tone.

- Uterus: position, shape and consistency, mobility, masses, pain or discomfort on palpation.

- Adnexa: size, contour and consistency of the ovaries, masses and discomfort on palpation.

- Fornices: rule out the existence of bulging and/or pain.

EXPLORATION TECHNIQUES

A. Examine the structures of the perineum.

1. Inspect the external genitalia:

This examination is of great interest, since by simply observing the vulva we can detect everything from the degree of sexual development of the woman if she is very young, to the presence of congenital malformations or some other previously undetected process.

mind.

You should observe the presence and arrangement of hair, the thickness of the large labia, the size of the clitoris, the presence of an intact hymen or not, vestibular secretions, the degree of vulvar opening and the presence or not of prolapse.

a) Before touching the genitals, touch the back of the woman's thigh with the back of your hand, to prevent sudden muscle tension with finger contact.

b) Then, use the index fingers and thumb of your non-dominant hand to gently separate the labia so that the clitoris, urinary meatus and vaginal opening are clearly visible (Fig. 20.7).

Note the color and pigmentation, the shape and symmetry of the structures, and whether there are any skin lesions or discharge, as well as the distribution of pubic hair.

Normal Findings: The color of the labia minora varies from pale pink to red. Brown or bluish pigmentation may be observed. The skin surrounding the lips should have pink or brown overtones. The labia majora are usually symmetrical and the shape of the labia minora ranges from triangular to semicircular. The edges of the labia minora may be smooth or irregular and may protrude through the labia majora.

Normal vaginal discharge is odorless, non-irritating, whose appearance varies according to the menstrual cycle.

After menstruation, a light whitish discharge may be observed; During ovulation there is usually a thinner clear secretion. After ovulation the secretion may again be thicker and whitish.

Some women observe changes in discharge or color and consistency of cervical mucus to estimate ovulation for pregnancy or contraception purposes.

2. Palpate Skene's and Bartholin's glands:

a) While continuing to separate with your non-dominant hand, insert the index finger of your other hand into the vagina. Palpate Skene's glands, applying light pressure against the anterior wall of the vagina and moving your fingers from the inside out (Fig. 20.8).

Secretion from the urinary meatus with this maneuver is abnormal and must be cultured.

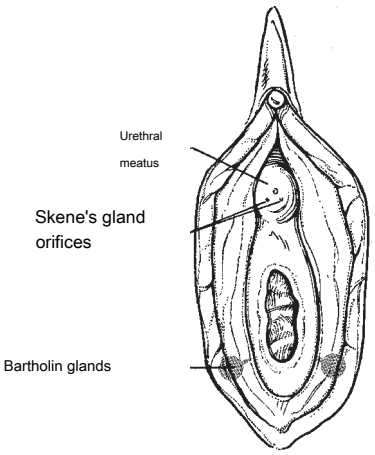
b) Palpate the Bartholin glands, on each side of the posterior vaginal introitus, placing your index finger inside the vagina, on the lower portion of each lateral surface, and your thumb in opposition, on the labia majora on that side. Gently press the skin between your thumb and index finger (fig. 20.9).

The appearance of discharge or pain indicates inflammation of the Bartholin gland (Bartholinitis).

Fig. 20.7 Inspection of the genitals.

Fig. 20.8 Palpation of Skene's glands.

Fig. 20.9 Palpation of the gland Bartholin right.



3. Examine the vaginal muscles:

a) With your index finger inserted 2-4 cm into the vagina, ask the woman to squeeze around your finger, to assess muscle tone.
Good muscle tone is manifested by the ability to narrow the vagina around the examiner's finger. Muscle tone is usually firmer in nulliparous women.

b) Place your index and middle fingers on the lower edge of the vaginal opening and separate the labia majora, moving the fingers laterally (fig. 20.10).
Ask the woman to push so you can inspect the vaginal opening. Look for any bulging, which may indicate a cystocele (herniation of the bladder into the vagina) or rectocele (herniation of the rectum into the vagina) or both (rectocystocele), and any leakage of urine.

B. Perform the speculum examination.

Fig. 20.10 Examination of the vaginal muscles.

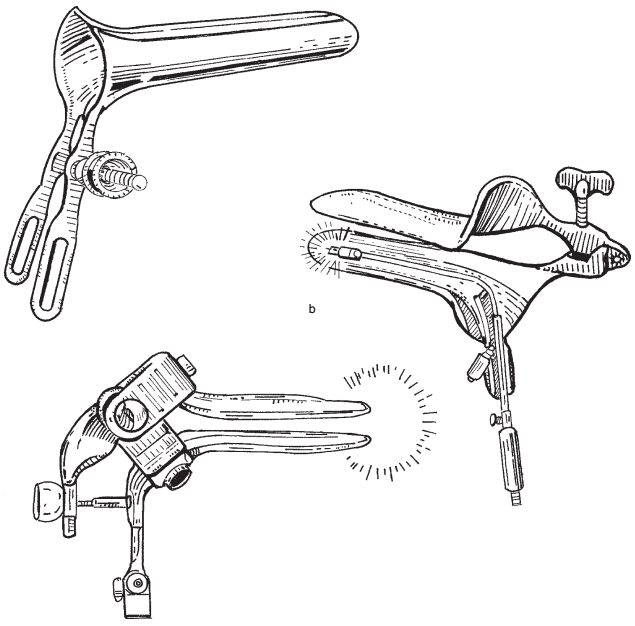
This examination is mandatory in the non-virgin patient and must be performed before the bimanual examination.

After inspecting the vulva, proceed to place the vaginal speculum.

Vaginal specula. They can be made of metal or disposable plastic, although the latter is usually more uncomfortable. They vary in shape and size. Graves specula are somewhat larger than Pedersen specula, which are used if the vaginal opening is very small (Fig. 20.11).

The speculum has two blades that separate to open the vaginal opening and a leveling or screw device to open and close the blades. Experience in its use should be gained by practicing opening and closing the blades before using it on a woman for the first time.

Fig. 20.11 Types of speculum: a, simple vaginal; b, with electric light; c, plastic material.



Vaginal specula are used to inspect the vagina and cervix.

By placing the speculum, you will be able to notice: the elasticity and length of the vagina, presence or absence of a foreign body (such as an intrauterine device, IUD), characteristics of the cervix, cervical processes and characteristics of the vaginal contents.

In addition, the speculum allows the performance of other investigations such as: Schiller test, vaginal or cervical scraping cytology, cervical mucus filance, colposcopy, vaginal exudate, etc.

Before inserting the speculum, you should warm it by running warm water over it or by storing it on a stove or heater at a low temperature.

A woman's natural vaginal secretions are usually sufficient lubrication to allow insertion of the speculum. If additional lubrication is necessary, the blades should be immersed in running water that is not cold. Vaginal creams, jellies, and lotions should not be used to lubricate the speculum, because they interfere with the collection of samples for analysis of secretions. Furthermore, such substances can irritate vaginal tissues.

1. Insert the vaginal speculum:

a) Open the vaginal opening by placing your index and middle fingers just inside the lower vagina and gently press down (fig. 20.12).

You can also separate the labia majora (in their middle part) with the other hand and insert the speculum arranging the width of the tip of the valves in an anteroposterior direction; or you can depress the perineum and pull it back to open the introitus.

In some multiparous women, the introduction is very easy and does not require any of the maneuvers described above.

b) Direct the blades or leaflets of the closed, pre-warmed speculum over your fingers into the vagina at a 45° angle, following the natural contour of the posterior vaginal wall. Insert the leaflets obliquely to minimize discomfort.

Be careful not to scratch or prick the genital tissue or pull the pubic hair. Insertion may be difficult if the woman has tight pelvic muscles. If this occurs, stop advancing the speculum momentarily and remind the woman to stay relaxed. Encourage her to breathe slowly and deeply and exhale through her mouth, through slightly parted lips, to relax and try to push. When this is reached, and when the vaginal introitus opens, continue advancing the speculum.

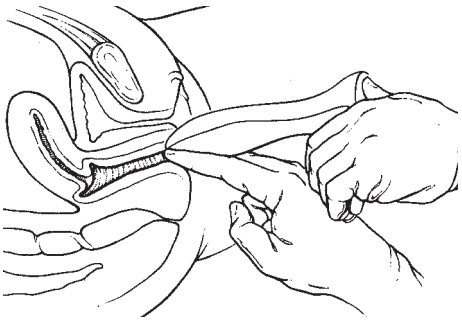


Fig. 20.12 Insertion of the vaginal speculum.

c) When the speculum is in place, remove your fingers, keeping the lower vagina open, and rotate it so that the leaflets are oriented horizontally. Actuate to partially open the leaflets by pressing the speculum elevator with your thumb.

d) Look through the open valves for the cervix. The neck will be completely visible if the leaflets are well positioned in the anterior and posterior sacs. If the neck is not fully visible, close the leaflets, withdraw it slightly and insert it again, at a slightly different angle.

e) Once the neck is clearly visible, fix the leaflets in the open position by adjusting the adjusting screw or nut with the thumb (metal speculum) or by fully depressing the elevator (plastic speculum) (fig. 20.13).

2. Inspect the cervix:

Look through the open leaflets to inspect the neck. Adjust the external light source, if necessary.

Shape and position: the neck is a rounded structure 3-4 cm in diameter, which protrudes about 2.5 cm into the vagina; Its position is determined by the configuration of the uterus. For example, in most women the neck is directed posteriorly. Without em-

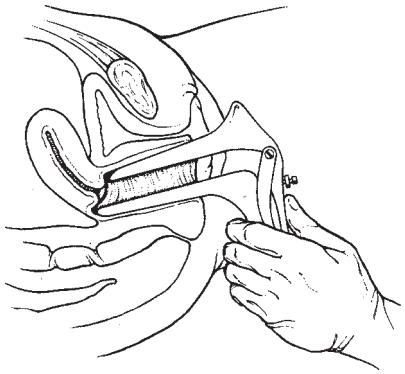


Fig. 20.13 Vaginal speculum placed in correct position.

However, when the uterus is retroverted, the cervix is directed anteriorly. The neck usually appears in the midline, rather than displaced laterally. The cervical opening appears as a small rounded depression in the nulliparous woman and as a flat indentation in the parous woman (Fig. 20.14).

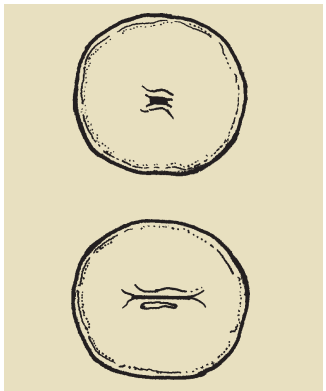
Color: It is usually pink and appears paler after menopause. In pregnant women, bluish pigmentation usually occurs around the sixth week. Oral contraceptives can cause dark pink to reddish cervical pigmentation.

Injuries: Because the neck can be torn during childbirth, the multiparous woman may have cervical scars from healed lacerations. Cervical lesions may indicate serious illnesses or infections and should always be carefully evaluated.

Naboth cysts, a relatively benign lesion, appear secondary to obstruction of glandular ducts. These cysts can appear in groups, as small yellowish papules, less than a centimeter in diameter. A guidewire coming out of the snout usually indicates an intrauterine device (IUD) used for contraception.

Discharge: A colored, bloody or purulent discharge is always abnormal.

Fig. 20.14 Appearance of the cervical os: a, in the nulliparous woman; b, in the multiparous.



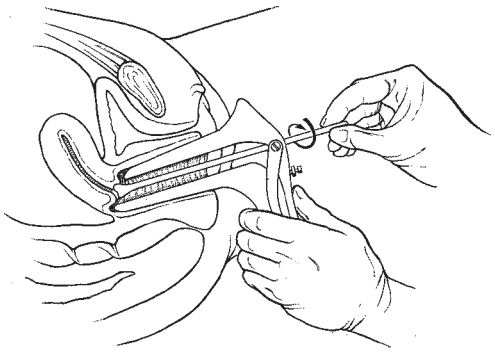


Fig. 20.15 Cervical scraping technique.

3. Obtain cervical samples (optional):

Three techniques can be used to obtain samples:

a) The cervical scraping technique. It involves inserting a specially designed wooden spatula through the leaflets of the open speculum. Place the spatula against the neck and scrape by rotating the instrument 360° against the cervical surface (fig. 20.15).

Remove the instrument and gently spread both sides of the end of the spatula on a glass sheet. Spray the sample with a fixative solution.

b) The endocervical exudate technique (fig. 20.16).

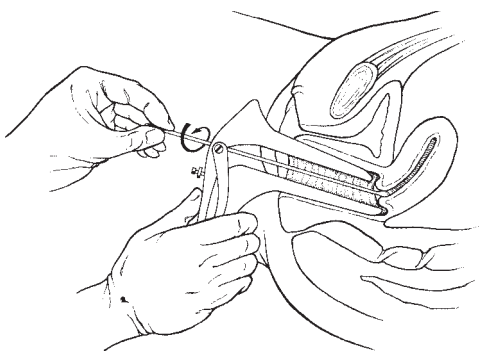
It consists of inserting an applicator with sterile cotton at the tip, through the leaflets of the speculum.

Insert the tip of the applicator through the cervical snout, about 0.5 cm. Rotate the applicator with your fingers 360° and hold the tip of the applicator in place for several seconds to allow saturation.

Remove the applicator and gently brush the sample onto a glass sheet, crossing from side to side, then spray the fixative on it.

c) The vaginal fundus exudate technique. It is done with the wooden spatula. Insert the spatula through the leaflets of the speculum into the posterior cul-de-sac. Scrape this area. Remove the spatula. Transfer the material to a glass sheet and spray it with the fixative.

Fig. 20.16 Endocervical exudate technique.



Cervical samples may be obtained for cytological examination (Pap smear), pathogen identification (gonococcal culture), or for fertility examination (evaluation of postcoital cervical mucus).

4. Inspect the vagina:

When you need to explore the anterior and posterior vaginal walls, rotate the closed speculum and open it laterally (at a 90° angle to the normal position) or insert it and open it later, without rotating.

You can inspect the walls of the vagina as you remove the vaginal speculum (Fig. 20.17). Release the locking device that holds the speculum open, taking care to manually hold the speculum open as you begin to remove it. Once the neck is no longer visible, allow the speculum to close slowly, so that the leaflets are completely closed when the speculum is exiting through the vaginal opening.

Removing the partially opened speculum may cause pain or pinching of the tissue if the leaflets suddenly close at the vaginal opening.

Normal vaginal appearance: it is pink in color with wrinkles, ridge-like structures, produced by folds of the mucous membrane.

Normal vaginal secretions can give the skin a moist, shiny appearance.

In postmenopausal women, the walls may be pale pink and less rough.

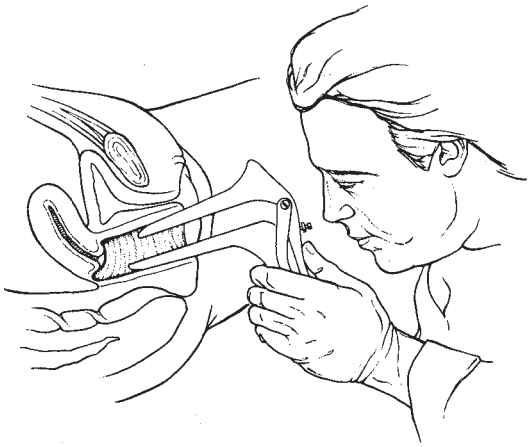
C. Perform vaginal examination.

After removing the speculum, proceed to perform the vaginal examination, for which you will use a sterile glove, preferably with lubricant.

1. Palpate the vagina and neck:

a) Stand in front of or to the side of the woman, whichever is easier for you, and gently insert the index and middle fingers,

Fig. 20.17 Vaginal inspection when removing the speculum.



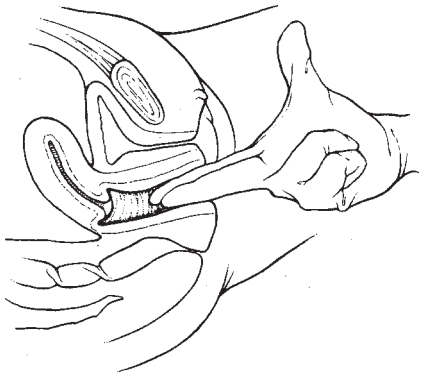


Fig. 20.18 Vaginal palpation.

gloved and lubricated, to palpate the vagina (fig. 20. 18).

b) Insert your fingers following the natural contour of the vagina, applying slight posterior pressure.

Keep your thumb abducted and the other fingers flexed. If the vaginal opening is very small, you will probably need to use only one finger.

c) Palpate the vaginal wall looking for nodules, masses or pain. Palpate the neck and determine its position, mobility, consistency and sensitivity.

Palpating masses is abnormal. Do not misinterpret roughness by masses. The neck is firm, similar to the tip of the nose, and mobile.

2. Perform bimanual palpation of the pelvic structures:

a) Stand with your index and middle fingers in your vagina.

b) Place the opposite hand, which may now be ungloved, on the abdomen between the navel and the symphysis pubis (fig. 20.19).

c) Use both hands to grasp the different structures that make up the internal genitalia and study their position, size, shape and consistency of the neck.

Fig. 20.19 Bimanual palpation.

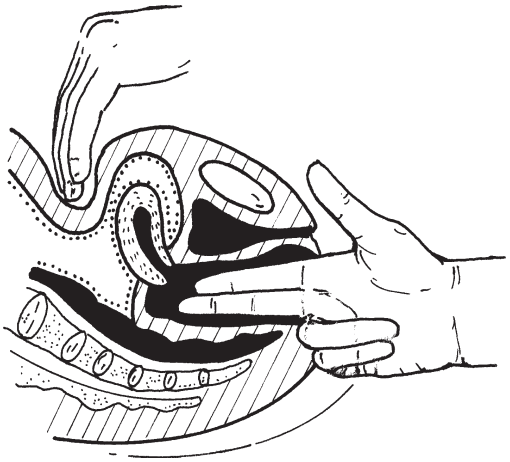


Fig. 20.20 Vaginoabdominal palpation of the uterus.

and the body of the uterus, thickness and length of the tubes, mobility and size of both ovaries, elasticity of the cul-de-sac and degree of painful sensitivity, as well as the presence or absence of tumors.

d) Palpate the uterus by pressing down with the abdominal hand directed towards the vaginal hand, which firmly supports the vagina, exerting light pressure against the perineum with the fingers that are flexed outside (fig. 20.20).

The uterus should be palpated just above the symphysis pubis and should be slightly movable when pressure is applied during bimanual palpation. On palpation, slight discomfort secondary to muscle tension may be noted.

The retroverted uterus is usually not palpable with this procedure.

Palpate the anterior wall and fundus of the uterus for masses or pain.

Normal findings: The uterus is pear-shaped and firm and smooth. The average size of the uterus of a non-pregnant woman is about 8 cm in greater or longitudinal diameter, of which 3 cm correspond to the neck; about 4-5 cm in transverse diameter at the level of the fundus (from horn to horn), which progressively decreases towards the neck, and about 3 cm in thickness or anteroposterior diameter.

e) Move your vaginal fingers toward the right lateral cul-de-sac and rotate your hand so that your palm faces up. Move the abdominal hand toward the right lower quadrant. The ovary and adnexa, which are not always palpable, can now be caught for examination, between your two hands (Fig. 20.21).

f) Now move your vaginal fingers towards the left lateral fornix and your abdominal hand towards the left lower quadrant for palpation of the left ovary and tube.

Normal Findings: The fallopian tubes are not usually palpated. The ovaries may or may not palpate

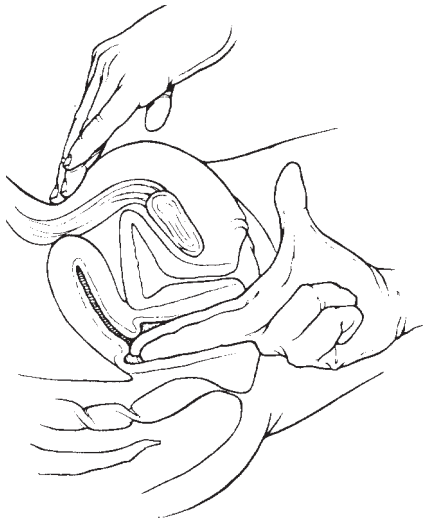


Fig. 20.21 Bimanual palpation of the right ovary and adnexa.

parse and may feel small, firm, almond-shaped, moveable and smooth, without masses. Its dimensions are: 3-5 cm in greatest diameter, 2-3 cm in transverse diameter and 1-2 cm in thickness. Slight discomfort on palpation is common.

The result of this examination will be noted in the clinical history and any modifications found will be drawn on it.

D. Perform digital rectal examination.

1. Take your fingers out of the vagina and change the glove.

Changing gloves protects against possible contamination of the rectum with vaginal secretions.

2. Lubricate the index finger of your newly gloved hand.

Explain the procedure and ask the woman to begin relaxation breathing, since deep breathing helps relax the anal sphincter.

Tell him that he may also feel the sensation of defecating.

The rest of the digital rectal examination technique was already described in the study of the digestive system, in Chapter 16.

Rectal examination is the logical choice in virgin women and is very useful in neoplastic processes, to detect the degree of infiltration of the parametria or uterosacral ligaments, as well as cystic tumors or not.

The indication of an evacuation enema, before performing this examination, is very useful, especially in constipated patients.

E. Complete the physical examination.

When you finish performing the scans, take your fingers out and clean the perineum with back-and-forth movements. Help the woman out of the stirrups and provide her with enough additional material to clean her perineum.

The examination techniques to detect or examine the evolution of pregnancy are set out under the title

of Increase in volume of the uterus", in Section II of Volume 2.

E EMPLOYMENT OF RECORDING THE RESULTS OF A NORMAL EXAMINATION

External genitalia and vagina moist, pink, without lesions or secretions. No alterations on palpation of the Bartholin's Skeney glands. Good tone pelvic muscles. Cystocele or rectocele not visible. Cervical orifice of nullipara, pink neck, in the midline, with little clear discharge. An endocervical sample is taken for vaginal cytology (Pap smear). Bimanual palpation does not reveal masses or pain. Uterus in the midline, mobile and anteverted.

CLINICAL PROPEDEUTICS. PATHOLOGICAL NOMENCLATURE. DISEASE, SYMPTOMS, SYNDROME, DIAGNOSIS AND FORECAST

21

CLINICAL PROPED UTICA

Years ago the subject General Pathology was taught in our Faculty of Medicine. In reality, given the great extension that many of the parts that composed it had acquired, such as Clinical Laboratory, Pathological Anatomy, Radiology, etc., the subject was limited to the study of symptoms, which is why it was also known by the name Semiology.

For many authors, General Pathology, Semiology and Clinical Propedeutics are similar, and with these names, in the curricula of the different universities, subjects whose content was the same were designated.

We, without entering into sterile discussions, want to establish the criteria we have of Clinical Propedeutics and also outline its content.

CONCEPT

Clinical Propedeutics is the study of the preparatory knowledge necessary for clinical teaching, or preliminary clinical knowledge as it has also been called.

In Clinical Propedeutics we will study two fundamental parts: Semiology and Syndromology.

Semiology, or more properly semeyology, given its Greek origin, is a compound word (semeyon: sign; logos: discourse), which means the study of signs, that is, according to its name, this part of Clinical Propedeutics studies all those phenomena that by their own nature or by simple convention evoke the idea of disease.

Syndromology, also according to its meaning, is the study of syndromes. We understand by syndrome a group of symptoms and signs that appear forming a clinical group that gives it individuality, but that can be due to multiple causes. For example, jaundice syndrome, hemolytic syndrome, etc.

We should not confuse it with a disease or nosological entity in which the symptomatic set is due to a single cause.

BRIEF HISTORICAL ACCOUNT

The history of semiology dates back to ancient Greece (5th century BC) and the medical school of HipŐcrates, considered the father of medicine and the founder of semiology, emerged in Cos.

He maintained that diseases could be recognized by the doctor's senses. It gave special significance to the observation of events and the exploration of the patient as a person.

The Hippocratic doctors were general practitioners and placed great importance on the doctor-patient relationship. The experience derived from caring for many patients led them to the configuration of a primitive clinical method with elements of anamnesis, inspection, palpation, as well as the elaboration of a diagnostic thought, the formulation of a diagnosis and therapeutic behavior.

The Hippocratic ethical code included in his famous "Oath" still serves as the basis for medical ethics. After the long medieval period in which no significant progress was made in medicine, we are witnessing a revival of the clinical method and semiology with Thomas Sydenham (1624-1689) who advocates returning to Hippocrates and practicing the profession at the bedside of the sick. He asserts that the doctor's duty consists of identifying the morbid species from the symptoms of the patients.

It was the Austrian Leopoldo Abenbrugger (1722-1809) who introduced percussion as a method of exploration in 1761 and the Frenchman René Teófilo Jacinto Laennec (1781-1826) introduced the method of mediated auscultation, using the instrument stethoscope (from stetos = thorax and esscopei = see, examine); This is the device for examining the chest.

From both contributions, physical signology acquires progressive relevance, especially in the study of diseases of the respiratory system, especially pulmonary tuberculosis, a true scourge of the time.

The correspondence of the symptoms that the patient presented in life with the autopsy findings gave rise to the anatomoclinical method promoted by Morgagni that founded a new anatomy and physiology and strengthened the scientific approach on the material origin of the disease and the method to identify it: from the symptom to the disease. On this aspect, Coste published a monograph years later that he titled: From the symptom to the disease. It is no longer mandatory to wait for the autopsy of the patient to know the injury that affected them, since knowing the symptoms and signs produced by it makes it possible to infer its presence.

With the contributions of Claude Bernard (1813-1878), who introduced the experimental method in scientific research, progress was made towards a pathophysiological interpretation of morbid processes, while following the discoveries of Luis Pasteur (1822-1895) in France and Robert Koch (1843-1910) in Germany, etiopathogenic medicine crystallized, formulating the bacterial theory of the disease.

Thus, at the end of the 19th century, the clinical method was formed in a finished form, whose application throughout the 20th century has achieved full development and verification with the extraordinary scientific contributions that have allowed clinical medicine to become a science.

Paradoxically, the current conflict lies in the undermining of the clinical method for the sake of technological apparatus, which calls for restoring harmony between technology and good clinical practice, on the principle that it is not an end in itself but a means that the latter uses to continually improve care for the health of human beings.

The study of symptoms should be carried out following, whenever possible, the system detailed below:

- Concept or definition.
- Semiogenesis or pathophysiology.
- Semiotechnics.
- Semiography.
- Semiodiagnosis.

Concept definition. It is the clarity and accuracy with which the generic and differential characteristics of the symptom are described.

Semiogenesis or pathophysiology. It is the study of the genesis or origin of the symptom. This part is perhaps the most important part of semiology and is also known by the names of pathophysiology and production mechanism.

It is on the occasion of semiogenesis that the need for a semiology of the normal arises, not only because illness is the polar opposite of normality and health, but because between the two there is no net and precise limit, but rather a dark zone of interferences and penetrations that vary from individual to individual and from one moment to the next in the different stages of life.

We will see later when dealing with symptoms in particular, how the study of the causes and mechanisms of symptoms and signs takes advantage of and benefits from the knowledge of normal and pathological human biology, the exact sciences, physics and chemistry and, finally, philosophical thinking and reasoning; in a word, of the integrity of human knowledge

hands.

Semiogenesis or pathophysiology thus comes to establish the universal laws to which morbid manifestations conform, considered in the abstract, and the adaptations and modifications to which the laws must be subject, when they refer to and apply to the concrete fact of the patient, who never loses his individuality, since both in illness and in health, he preserves his personality and imprints his own modalities on it.

Let's take a simple example: when we talk about renal edema, it is convenient to know that it is due to sodium and water retention, determined by a decrease in the oncotic pressure of proteins (intense hypoproteinemia) that causes the passage of fluid from the intravascular to the intercellular space; This determines an increase in plasma osmolarity that stimulates the hypothalamus and this in turn, through the neuroendocrine route (production of antidiuretic hormone and aldosterone), acts on the tubulus renalis to determine greater sodium and water retention.

Semiotechnics. It consists of the methods, procedures, resources and techniques that we use to obtain the symptoms.

Classically, anamnesis or interrogation, inspection, palpation, percussion and auscultation have been used for these purposes. At present, a series of auxiliary examinations and examinations are also used to obtain data of fundamental value for the diagnosis. For example: a pneumonic focus located more than 6 cm deep in the lung parenchyma is not detectable by classical methods, however it is easily accessible by a fluoroscopic or radiographic examination.

Semiography. Also called clinical study or characterology; It studies the description or characteristics of the symptoms and signs, the only way to give them a classification that makes them useful for diagnosis and prognosis.

Indeed, the simple statement or recognition of a symptom by one of its most objective characteristics, such as, for example, cyanosis by its color, does not in itself have greater semiological significance, since this sign is common to a countless number of diseases. But if when describing cyanosis we point out that it is very dark, that it is observed in the neck, head and upper extremities and is accompanied by venous stasis in those regions, then we will have obtained from this characterization a cardinal sign of mediastinal venous compression.

Another example: in the study of epigastric pain we must describe location, irradiation, periodicity, rhythm and schedule, intensity, character, duration, evolution, accompanying symptoms, etc.

Semidiagnosis. This term is synonymous with semiological value or semiological significance. We found it more useful to create this term, because it maintains greater uniformity with those previously indicated.

It generally refers to the value that the symptom has for the diagnosis of a disease, or, if preferred, all those diseases in which the studied symptom is present can be considered here.

From this concept it follows that, sometimes, a symptom can serve to guide towards a diagnosis, and in others, however, it is common to several diseases, which is why we have to use the set of symptoms grouped into syndromes to diagnose the disease. For example, knowing that the existence of a diastolic drag with presystolic strengthening and with a click of opening of the mitral valve is the auscultatory expression of mitral stenosis.

PATHOLOGICAL NOMENCLATURE

It is important to know, at the very threshold of pathological studies, what the foundations of its nomenclature or language are. It has been said, quite rightly, that to know a science is to know its terminology.

What is missing in semiology is the existence of a true pathological nomenclature that, as in chemistry, somewhat orders its terminology. It can be said that the names with which diseases are designated have not been, nor practically are, subject to any rule or convention, which is perhaps explained by the slowness with which pathological science has been formed.

The origin of the names of diseases is surprising for its diversity. Sometimes it depends on the similarity that is discovered to the disease with this or that; others, of its cause, duration, location, color, age at which it appears, etc.; Others, finally, bear the name of the doctor who described it for the first time, or of the one who described it in the most complete or attractive way.

Thus, cancer took its name from the resemblance it bears to the crab; Phlegmasy and fever translate the idea of burning or boiling; consumption, that of consumption or extreme emaciation, and chorea, that of dancing or dancing. Lead poisoning owes its name to the cause that determines it: lead poisoning; the same as argyrism or hydrargyrism, poisoning by silver and mercury, respectively. The name of the diseases scarlet fever, erythema and purpura depends on the color they determine on the skin.

Infantile paralysis owes its name to the predominant paralytic symptoms and the age at which it most frequently appears. Pleurisy and hysterism originate at the supposed site of the disorders: the pleura and the uterus, respectively. Many names of diseases constitute a tribute, well deserved when well justified, to the clinicians who described them, such as Vaquez-Osler's disease and Recklinghausen's disease; Fröhlich syndrome, etc.

However, some disease names are formed using known suffixes and prefixes, according to certain rules, the meaning of which characterizes the disease.

Among the most used suffixes are: itis, which means inflammation (peritonitis, pleuritis, arthritis, etc.); omna, which corresponds to a tumor (epithelioma, sarcoma, hypernephroma, etc.); ectasia, indicating dilation (gastroectasia, typhloectasia, bronchiectasis); cele, meaning hernia (meningocele, enterocele); ragia, which expresses blood loss (hemorrhage, gastrorrhagia, enterorrhagia, stomarrhagia); Rhea, which corresponds to flow (sialorrhoea, gonorrhea); algia and odinia, which translate the existence of pain (gastralgia, appendicalgia, pleurodynia): plegia, which means paralysis (monoplegia, hemiplegia, paraplegia); oid, indicating similarity (diabetoid, leukemoid); ism, which also indicates similarity (meningism, peritonism).

Among the prefixes we can mention: pseudo, which expresses the idea of falsehood (pseudotabes, intestinal pseudoocclusion, pseudoappendicitis); peri, meaning around (periarthritis, peribronchitis, pericholecystitis, perigastritis), and para, meaning beyond (paraphimosis, paraphrenia).

The usefulness of regulating pathological language is indisputable. If on the one hand the advantages of a well-regulated pathological nomenclature are obvious, on the other, the experience acquired with some efforts made for this purpose indicates that the current defects constitute a much lesser evil than that which would be produced by trying to replace the classical denominations.
Furthermore, the lack of knowledge that still exists about the nature and causes of many diseases would always be a serious obstacle in the case of regulating pathological terminology.

DISEASE

Multiple definitions, both complex and incomplete, have been proposed for the disease. With Novoa Santos we believe that it is easier to arrive at this concept starting from the phenomena that take place in the state of health.

Man - like animals - can be conceived as a system that entails a series of reactional possibilities. It is capable of reacting to the most varied and new stimuli, in which it essentially differs from an automaton, whose responses are predetermined.

These reactional possibilities with which the organism is endowed undoubtedly give it a capacity for adaptation, exercised essentially by the nervous system and cellular metabolism. The capacity for adaptation allows the physiological activities of the healthy organism to oscillate within fairly wide limits, as proven by the fact that they can cope, in a certain magnitude, to the most varied demands of work and living in the midst of the continuous variations of the external environment, without breaking "that particular state of balance that we commonly designate with the expressions: state of health, sanity or hygienic state.

The state of health is, therefore, accompanied by a series of functional oscillations that we call physiological phenomena and, subjectively, it is translated by a general feeling of well-being, the feeling of health, or euphoria.

As is understood, the organism's capacity to adapt is restricted and its accommodation to the different stimuli and circumstances it faces is limited by that restriction.

When the new conditions to which the organism is subjected cross the limits compatible with the maintenance of the physiological state, functional and organic disturbances will occur expressive of a particular state of maladjustment that constitutes the disease.

Disease, therefore, must be considered as a process that reflects the lack of adaptation of the organism to the most varied morbid agents (from Latin, morbus: disease).

Just as the state of health is translated by a series of physiological phenomena and a subjective sensation of well-being or health, the disease is manifested by the appearance of a series of abnormal phenomena, either susceptible to being picked up by the observer, or only accessible to the examination of the patient himself, who can experience sensations, of a very diverse nature, that indicate his illness, integrating a feeling of illness exactly opposite to the feeling of health to which we allude. before.

symptoms

These pathological phenomena, objective or subjective, that accompany the disease, that "fall with" the disease, are called symptoms (from the Greek, symptoma, from the verb sympipto: to fall with, to fall together with another).

Symptoms are, therefore, the phenomena of the disease, its ostensible manifestations, its expressive forms, as Letamendi called them, according to Galen, following the disease like shadows.

The symptoms accessible only to the introspective examination of the patient are called subjective symptoms; while those that can be found through medical observation are called objective symptoms.

We must note, now, that if symptoms are the most important signs revealing the disease, they are not the only ones, because signs are also, for example, the cause or course of the disease,

the age and race of the patient, the place of origin, his diet, etc. ; and it is that sign (from the Latin signum: signal, indication) in pathology, for us, is any phenomenon that can constitute a sign or indication of disease.

In the sign, there is, therefore, a judgment, a mental work. The symptom becomes a sign when we give it a revealing value of a certain disease.

Likewise, a personal data of the subject, an environmental circumstance, which the doctor values as an expression of any morbid process, becomes a sign. The concept of sign is, therefore, broader and more comprehensive than that of symptom. Therefore, according to Fernel, we can say that every symptom can be a sign, but not every sign is a symptom.

These conceptions of symptom and sign are not universally shared. English-speaking doctors consider any objective evidence of a disease a sign. For them signs are objective symptoms and they reserve the word symptoms for subjective ones. They also very frequently use the expression physical signs to indicate objective symptoms.

CLASSIFICATION OF SYMPTOMS

The importance of the symptoms and their variety demand a good classification.

We have already distinguished between objective and subjective symptoms. Objective symptoms or physical signs - the most important - are those that the doctor can collect through examination, such as a tumor, the case of edema, the dullness of a pleural effusion, the murmur of a heart valve lesion. Subjective symptoms are those that only the patient can perceive (headache and other pains, vertigo, tingling, cramps, lack of appetite, weakness, etc.). There are symptoms, however, that are both subjective and objective, such as dyspnea.

Symptoms can also be classified as general, local and distant.

General symptoms are those that express an alteration or disturbance of the entire organism, such as fever or hypothermia, weight loss, decay, etc.

Local symptoms are, on the contrary, those that are somewhat circumscribed, in close and direct relationship with the site of the disease. Among them we can mention the increase or decrease in volume of a viscus, inflammatory redness, crackling rattles of pneumonia, radiological signs of a respiratory (pulmonary) process, or another system, etc.

Remote symptoms are those that manifest in organs other than those in which they radiate. ca the disease. They can be reflex, toxic or hormonal, according to the mechanism that produces them. In reflex symptoms, the phenomenon takes place via the nervous system, such as the dilation of the pupil that accompanies certain processes of the pulmonary apex, or the vomiting of appendicitis, or the pain of the glans and testicle in renal lithiasis. Toxic symptoms depend on poisoning, such as vomiting in uremia, respiratory rhythm disorders in decompensated diabetes, in acidosis and in uremia itself. Finally, symptoms of hormonal origin are due to the action or lack of certain hormones secreted by the internal secretion glands. Among them we can mention the bone disorders that accompany eosinophilic adenoma of the pituitary gland (acromegaly), myxedema from hypothyroidism, hirsutism and hair loss from certain endocrine processes.

According to their diagnostic value, symptoms are classified as common and pathognomonic.

Common symptoms, also called indifferent, banal, equivocal and plurivocal, are those that can be found in various diseases, therefore, they are of very limited diagnostic value. These include anorexia, weakness, headache, vomiting, etc.

On the other hand, pathognomonic symptoms, also called characteristic or univocal, have great diagnostic value and denounce with their mere presence the existence of a certain disease. Strictly speaking, there are almost no pathognomonic symptoms; rather, certain symptomatic groupings or syndromes are considered as such. The opening click of the mitral valve upon auscultation in mitral stenosis of rheumatic etiology could be mentioned as a pathognomonic symptom.

Based on the moment in which they appear and the way in which they do so, prodromal or prodromal symptoms can be distinguished, which are those that appear early in the early stages of the disease they herald, so to speak. They are almost always imprecise. This is the case of headache in typhoid fever, weakness and low-grade fever in pulmonary tuberculosis. Sudden onset symptoms are called fits or attacks, and strokes or insults in Neurology. The period during which the symptoms reach their greatest intensity is called paroxysm.

Symptoms can be classified according to their evolution. Thus, in addition to distinguishing, as we have already done, those that appear slowly or suddenly, we must consider those that remain stationary throughout the evolution of the disease, or those that experience, on the contrary, oscillations of variable intensity (fluctuating symptoms), or those that disappear completely for

appear again (intermittent symptoms). Recurrent and intermittent fevers, respectively, can be cited as examples.

The study of the agreement between symptoms and morbid states is of great interest. First of all, it is necessary to establish that feeling sick, that is, the feeling of illness, is not present in all pathological processes, since there are many that are endured without any subjective discomfort, with the patient sometimes completely ignoring their true state.

These same sensations of illness do not have a constant relationship with the intensity and severity of the morbid process. Thus, we observe seriously ill patients whose feeling of well-being contrasts markedly with their deplorable state, while other patients, without any organic injury, suffer intensely, as a consequence of unfounded fears or worries that are the starting point of annoying psychic representations. These diseases that do not have demonstrable lesions have been called imaginary diseases. Therefore, it can be said, in agreement with Fleiner, that from the point of view of subjective symptoms each patient subjectively assesses the importance of their illness according to how they feel the magnitude of their illness, that is, they are as sick as they feel.

Many times it is observed that the patient lacks the sense of self-criticism that does not allow him to acquire awareness of the disease and a euphoric feeling is established that contrasts with the serious existing injuries. Thus, this morbid euphoria can be seen in the last periods of pulmonary tuberculosis, in the termination of numerous diseases and in the dying period, perhaps, as an expression of a true premortal psychosis. A feeling of exuberant health is also observed in manic attacks, in general paralysis and in tumors of the frontal lobe where it is combined with a certain joviality and tendency to joke and tell jokes, which constitutes a special mental state that has received the name moria.

From the point of view of the relationship between symptoms and anatomopathological lesions, we can say that the same symptom can be caused by different diseases, such as seizures, observed in brain tumors, encephalitis, epilepsy, uremia, etc.; such as the headache that can be observed in meningitis, brain tumors, cranial hyperostoses, etc. Therefore, the same symptoms can occur due to different diseases.

rents.

In turn, different symptoms can be observed in the same disease. A very demonstrative example is that of brain tumors whose symptoms vary according to with its location, although its histological structure remains the same.

There is also no constant direct relationship between the intensity of the symptoms and the importance of the lesions that produce them. In the same case of brain tumors, large tumors of the frontal lobe are seen to evolve almost without clinical symptoms, while others, much smaller, at the base of the brain, are extraordinarily rich in symptoms.

There are lesions that evolve without symptoms, they constitute the so-called latent diseases (latent tuberculosis, latent syphilis, latent diabetes). Other times, a specific disease presents itself to us with the symptoms and appearance of another: these are latent or masked diseases (tuberculosis with symptoms of typhoid fever, for example).

Many diseases evolve, from the point of view of their duration and symptomatology, typically: they are cyclical diseases, such as fibrinous pneumonia.
typhoid fever, etc. These evolutions have been modified with the use of antibiotics, for example. Others follow a very varied and irregular course, these are atypical diseases. Finally, some pathological processes may present mild symptoms of short duration, and are therefore called abortive diseases.

Very rarely do diseases manifest themselves with a single symptom (monosymptomatic diseases), they generally present with a set of symptoms, more or

less numerous.

SYNDROME

This name is known as the set of symptoms and signs that frequently appear always associated in the same way, but that may correspond to different etiologies. For example: jaundice syndrome with all its clinical characteristics can be caused by alterations in red blood cells (hemolytic jaundice), liver alterations (hepatocellular jaundice) and alterations in the bile ducts (obstructive jaundice).

DIAGNOSIS

The practice of so-called curative medicine, as Loewenberg states with accurate vision, rests on two fundamental bases: diagnosis and treatment. Of them, the first, and most important, is the diagnosis, since the institution of treatment depends on it.

Since diagnosis is, without a doubt, the cornerstone of medicine, mastering and knowing it must be the essential goal of every doctor. Its importance does not need to be highlighted after what we have said. When diagnosing

To diagnosis we will dedicate all our efforts and the better doctor will be the one who is best suited to carry it out and has the technical resources we have for it.

As we already said, the diagnosis - derived etymologically - from the Greek, day: through; gnignoskein: knowing- is the identification or knowledge of the disease, that is, the judgment by which the existence of a certain morbid process is affirmed that is distinguished from other morbid processes with which it may be analogous, and the disease is even known through the disguise with which it tried to hide, as Grande Rossi said.

To propose the provisional diagnosis we can use different procedures, which can be reduced to four main ones:

1. Diagnosis by intuition.
2. Diagnosis by comparison.
3. Diagnosis by reasoning.
4. Diagnosis by hypothesis.

DIAGNOSIS BY INTUITION

It is done by simple recognition of the set of characteristic symptoms of a disease, without any reasoning. It is very difficult, requires extraordinary experience (clinical eye) and is prone to errors, except in certain cases of very visible lesions, or when the data provided by all means of clinical examination are assessed at a glance. It is also called direct diagnosis.

DIAGNOSIS BY COMPARISON

The symptomatic picture of the patient is compared with that corresponding to the most similar diseases. It is a true differential diagnosis, as it is also named, in which the doctor compares the symptoms of the patient with those of the most similar diseases, mentally specifying the analogies and differences, until diagnosing that disease whose symptoms coincide almost exactly with those of the patient being studied. It is a very safe diagnostic procedure, especially when supported by a complete examination of the patient, and the most frequently used. There is a variety of diagnosis by less exact comparison called diagnosis by exclusion or by successive eliminations. Here we start from the organ or system that is supposed to be sick, and all the diseases it may suffer from are reviewed, excluding those that do not correspond to the patient's symptoms, leaving one unexcluded, which is the one that is diagnosed, even when there are not always powerful reasons to affirm its existence. Hence its lower security,

DIAGNOSIS BY RATIOCINI0

When the doctor, prevented from reaching a diagnosis by the previous procedures, due to not finding any disease whose symptomatic picture corresponds to that of the patient, reflects on the present symptoms, relying on his knowledge of physiology and pathological anatomy, and concludes by identifying a unique process that explains the existing disorders. It would be the method to follow in the study of an unknown pathological process, not described until that date. In reality, diagnosis by reasoning is also diagnosis by comparison.

DIAGNOSIS BY IPOTHESIS

Also called Hufeland therapeutic. The doctor, who has not arrived at any diagnosis, replaces it with a hypothesis, waiting to confirm or reject it on the subsequent course of the disease or the result of the treatment. Falling into this diagnostic procedure should be avoided by all means, and, in any case, if used, insist repeatedly on the observation and study of the patient to form a definitive diagnosis. The diagnosis by hypothesis can only be a provisional diagnosis. However, we must confess that sometimes, and despite the valuable cooperation provided by the most accurate diagnostic means, we have to settle for formulating one of these diagnoses by hypothesis, which the advance of medical science should have made disappear. And in reality, given the difficulties of the clinic, even the most complete diagnosis is nothing more than a diagnostic hypothesis, eminently unstable, and according to Nieto Serrano, the diagnosis is always being formed while the disease lasts.

In the diagnosis, these procedures will always be used, preferring those of comparison and reasoning, without disdaining the intuitive method, nor fearing the hypothetical diagnosis, which is often necessary, and even useful, when it is supported by a careful and repeated examination of the patient and is accepted as provisional with the firm intention of making all the necessary efforts to reach the final or definitive diagnosis.

It is convenient to begin with the investigation of the signs or symptoms (semiotechnics), then these are grouped and interpreted forming syndromes and looking for their cause (Clinical Propedeutics).

In a complete diagnosis, the following partial diagnoses must be made:

1. Diagnosis of symptoms and signs.
2. Diagnosis of syndromes (pathophysiological diagnosis)
3. Anatomical diagnosis.
4. Etiological diagnosis.
5. Diagnosis of functional capacity.

To do this, we can use fundamental clinical techniques: interrogation, inspection, palpation, percussion and auscultation, aided by auxiliary, instrumental and laboratory procedures that are often as essential as those.

The best examination method is the most complete and orderly one. The doctor has the duty to always carry out a comprehensive examination of the patient, regardless of the direction in which the symptoms collected in the interrogation guide him. According to Araoz Alfaro, diagnostic errors depend, for the most part, on incomplete, hasty or insufficiently repeated examinations, and in medicine there are no small signs or useless examinations.

Therefore, a good examination method must be adopted, which will be respected and followed faithfully in all cases, in which order and thoroughness must dominate, in addition to being complete as we have already said, without failing in any case to explore all the organs and systems, using all the examination resources. Regarding exploration resources, whether clinical, radiological or laboratory, we will say like Alfaro, that there are not and cannot be hierarchical oppositions or preferences, because they are all necessary and even, sometimes, insufficient for the diagnosis. Let us always remember, according to Sergent, that each examination method has its limit of sensitivity, that is, any of them, the one that may seem less accurate, is capable of providing signs that no other procedure, even the most precise ones, can provide.

There is no diagnosis without medical science and art in the broadest sense of the word. Science that is based on knowledge and art that is born from observation and practice.

Intuition and diagnostic divination, the "clinical eye" of good doctors is nothing other than "clinical knowledge", since knowing, according to G. Le Bon, is doing unconsciously what was consciously learned.

FORECAST

Prognosis (from the Greek, pro: ahead, in advance, and gnignostein: to know) is called the judgment given by the doctor about the evolution that an illness will have, that is, about its alternatives, duration, probable termination and sequelae.

Establishing the prognosis continues to be, as in the time of Hippocrates, the most difficult part of medical practice and also the most delicate; The anticipatory indications depend on it, whether therapeutic and/or psychological, that the doctor must institute and take for the safety and tranquility of the patient and his or her family.

It can be said that there is no other rule or method to make the prognosis than to make the complete clinical diagnosis of the patient, since this diagnosis he almost carries the prognosis within himself; The rest is done by some particularities of certain signs and above all by the art and experience of the clinician.

The forecast always raises these two problems:

Does the disease compromise the life of the patient?

What far-reaching consequences will it have?

According to this, the prognosis can be: benign, serious, lethal and reserved.

Benign prognosis. When the disease cures completely and quickly with complete restitutio ad integrum.

Serious prognosis. When the illness can have alternatives that endanger the life of the patient, when it is very long or painful, or when it can leave sequels or various conditions.

Lethal prognosis. When it is a fatal disease, either due to its severity or because there is no efficient treatment.

Reserved forecast. When among the alternatives that it may present are serious consequences and conditions and the possibility of death.

The clinical diagnosis, we said, in itself carries the prognosis, but this is "a future" and there is nothing more uncertain than the future in the life of the sick man. An insignificant wound, a trivial tonsillitis, can end in sepsis or serious and even fatal nephritis.

Pulmonary tuberculosis and typhoid are serious in themselves, and can leave sequelae, but they can also evolve benignly and heal completely. On the other hand, cancer, nephroangiosclerosis, chronic circulatory insufficiency, etc., will march, in a more or less short or long term, towards death. From these examples it is clear how much tact, moderation and knowledge are necessary to establish a forecast, which although it can be guided "by the general rule" the exceptions should not be forgotten.

When studying the examination method that should be chosen to more safely reach a complete diagnosis, the advice of Robert Hutchinson, reproduced by Levy Simpson in his interesting book on medical diagnosis, should be remembered:

1. Don't be too smart.
2. Don't diagnose oddities.
3. Don't be in a hurry.
4. Do not lose interest in the diagnosis.
5. Don't confuse a label with a diagnosis.
6. Do not diagnose two diseases simultaneously in the same patient.
7. Don't be too sure.
8. Don't be biased or biased.
9. Do not hesitate to revise your diagnosis from time to time in a chronic case.

PRODUCTION FACTORS AND MECHANISMS OF ILLNESS

22

PRELIMINARY CONSIDERATIONS

HEALTH AND ILLNESS

The World Health Organization (WHO) has defined health as the state of complete physical, mental and social well-being and not only the absence of disease. This definition overcomes the traditional one-dimensional organic concept of health and illness by also including the psychological and social dimensions. Its main limitation lies in the fact that it does not highlight the constant interaction of man with the environment, nor the continuous transformation of this and that.

We can consider health and illness as a dialectical pair relative to the balance, favorable or unfavorable, that man maintains with his natural and social environment.

ETIOLOGY AND PATHOGENESIS

There are different factors that, either in isolation or more commonly in association, can break this balance and cause the appearance of the disease (from the Latin, infirmitas: without firmness, without strength).

The causative or etiological factors (etiology: from the Greek, etio: cause; logos: study or treaty) can be classified into:

Fundamental factor or etiological agent

It is the isolated factor that predominates in the appearance of the disease.

Contributing factors

Among them we can consider:

Predisposing factors. They represent the biopsychosocial "terrain" of the subject, which makes him prone or particularly vulnerable to certain agents.

Triggering factors. They precipitate or put into action the chain of events that makes a previously obvious illness evident.

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Perpetuating factors. They are those that "entertain" the disease, hinder recovery.

Configuration factors. They contribute to the location or form of presentation of the morbid process.

Irreversibility factors. They unfavorably influence the evolution of the disease, conditioning its fatal outcome.

Pathogenesis (from the Greek, pathos: disease; genus: origin) does not study the factors causing the disease, but the functional and morphological mechanisms originated by those, which make up the cellular, tissue, humoral and clinical reactions of the patient.

Like the etiological factors, the pathogenic mechanisms are also usually multiple. The relationship between the etiological agent and the pathogenic mechanisms is so evident and difficult to distinguish that we often speak of etiopathogenesis. In certain processes (allergy, self-harm, etc.) it is difficult to unravel whether the essence of the disease lies in the action of the agents that appear as triggers, or if, on the contrary, the changes that have occurred in the body constitute its real cause.

MAIN AGENTS OR RISK FACTORS

The agents capable of triggering disease are very numerous. According to Roessle, there is not a single force among all those known, that act between heaven and earth, that in a certain intensity is not capable of causing a disorder in our organism; We consider that the list of etiological factors is as extensive as the nature of the universe itself.

In certain diseases it is easy to recognize that the causative agents or factors are external to the body.
This is the case of epidemic or infectious diseases, burns, etc.

Other times the disease seems to be related to internal agents or factors of the organism that suffers from it. For this reason we speak of exogenous, external or ecological etiological factors and of endogenous or internal etiological factors. The opposition between both groups of factors is more apparent than real, since every human being lives in an external environment that influences, at least in part, his internal environment, and, in turn, this will condition the action of the former.

The agents or factors that we will study next are (fig. 22.1l):

- Mechanical factors.
- Physical factors.
- Chemical agents.
- Biological agents.
- Genetic factors.
Immunological factors.
- Neoplasms.

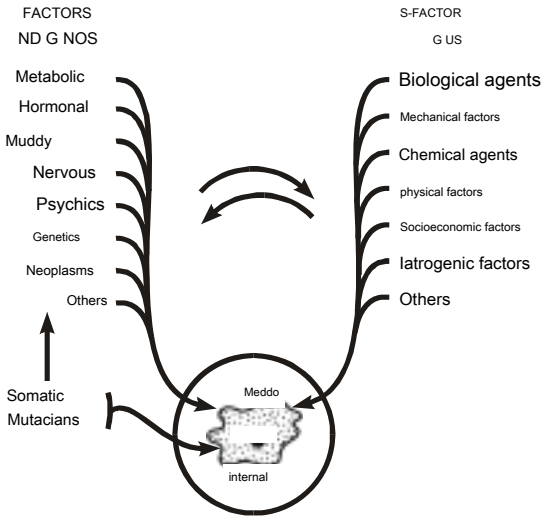


Fig. 22.1 Main factors or disease-producing agents.

- Psychological factors.
- Iatrogenic factors.
-Socioeconomic factors.

Since socioeconomic factors are those that, ultimately, condition or determine the rest, we are going to study them first.

SOCIOECONOMIC FACTORS

Nature of the human being

The epistemological study of health, illness and their determinants leads us to scrutinize the very nature of the human being. What is the essence of man:
Is it a biological or social being?

The correlation of the biological and the social in human determinism has been the object of study and debate at scientific and philosophical levels. Without attempting to delve into this field, we will only stick to what was stated by some philosophers who said that man is, in his immediate form, a being of flesh and blood and, however, his essence is not biological, but is given by the set of social relations. By acting through work on abstract nature, man distinguishes himself from the animal kingdom.

Transforming the nature outside him, he transformed his own nature and the relationships that man established with other men in the process of production gave rise to consciousness, a specifically human trait.
hand.

The definition of man as a social being in no way dismisses his biological aspect, which represents his starting point. The dialectic of the forms of movement of matter clarifies that the highest forms arise historically from the lower forms.

These are not eliminated, but are subordinated and transformed by those that contain them.

In the same way that the physiological level includes and surpasses the biochemical level, without eliminating it, the social level, which represents the higher form of movement of matter, contains the biological level, which is the form that immediately precedes it. From the reciprocal relationship between both forms, the psychic phenomenon, also socially conditioned, emerges as a link.

Socioeconomic structure and health-disease process

Like man himself, the health-disease process has a double character: biological in its form and social in its content. There are biological laws that influence the development of man and the determinism of his state of health, but they are subject to social laws, which, ultimately, are decisive.

The historical experience of our country provides convincing evidence of the determining nature of the mode of production in the state of health of the population. When unemployment, hunger, misery, ignorance and medical-health neglect disappeared, a notable transformation occurred in the morbidity and mortality of the population. Communicable diseases have been eradicated or drastically reduced (such as diphtheria, tetanus, whooping cough, malaria, poliomyelitis, gastroenteritis, among others), as have nutritional deficiencies.

These diseases have disappeared from the top ten causes of death. Mortality in especially vulnerable groups, such as pregnant women (maternal mortality) and infants (infant mortality), has experienced impressive reductions. The life expectancy of the population has increased significantly.

Suprastructure and health-disease process

Changing the economic structure of society, in which exploitation and social inequality disappear, does not automatically eliminate all problems.
Although this change in the macrosocial sphere determines changes in the microsocial relationships between people (family, school, work, community environment), these do not occur immediately, simultaneously, or spontaneously.

The suprastructural factors that make up social consciousness influence the health-disease process to varying degrees. Such factors are, for example, religious beliefs, food taboos, cultural and moral patterns, and habits and knowledge related to health.

From a health culture inherited from the past, certain lifestyle habits persist in our population that have been revealed as true factors that cause disease. These risk factors include smoking, a sedentary lifestyle, excessive consumption of nutrients (carbohydrates, fats, salt), and psycho-emotional tension. These cultural patterns and adverse microsocial relationships can contribute to the origin and development of serious diseases such as various neuroses and psychopathies, accidents, peptic ulcer, high blood pressure, obesity and diseases dependent on arteriosclerosis, among others.

Socioeconomic development and its impact on the health-disease process

The social conditionality of human pathology indicates the only possible way to combat it: to produce transformations in the macrosocial structure, followed by suprastructural changes, and in microsocial relations so that they contribute, together with the development of science and technology, to develop and perfect the social environment of man.

In the future will man be able to get rid of the diseases that affect him today? Could he then live healthy or would new diseases appear?

In human nature there is no foundation that allows us to sustain the deeply rooted idea that illnesses constitute an unalterable fatal necessity.
Diseases appear and disappear, rise and decline, in relation to a certain socio-economic formation.

R. Virchov, creator of cellular pathology and one of the founders of social medicine, reflected on the matter: "Isn't it true that mass diseases everywhere point to deficiencies in society? Atmospheric or cosmic states or similar factors can be adduced. However, they are never the cause of epidemics.
They occur only where, because of bad social circumstances, people have lived for some time in abnormal situations. Epidemics of a hitherto unknown character appear and often disappear without leaving a trace when a new cultural period has begun. This is what happened with leprosy and with the English sweat. Consequently, the history of artificial epidemics is the history of disorders of human culture. Its changes announce to us with gigantic signs the points at which culture turns in new directions. Epidemics bear similarities to great warning signs, in which the true statesman can read that the evolution of his nation has been disturbed, to the point where he cannot even afford careless policy."

Achieving full well-being and the free and total development of all members of society is usually considered

a utopia in today's world. Perhaps we will have to wait for humanity to achieve such a degree of material, cultural, spiritual and moral development that allows it to jump from the realm of necessity to the realm of freedom, in a new conception and praxis regarding the relationships of men with nature and with each other, which protects and promotes the life and health of the planet and all its inhabitants.

MECHANICAL FACTORS

Mechanical movement can produce harmful effects, both by action and by omission. The first case is represented by mechanical violence and the second, by immobility and sedentary lifestyle. From now on we draw attention to the social 'conditionality' of both.

Mechanical violence

Mechanical violence causes bodily injuries called trauma, which are produced by the collision between an object and the human body;

you may be interested in:

art ea {Fractures

i Contusion

art la da 1

¡ Blunt í

1 lost

Incised î

Sharp

rga o i ter o

ìSolid viscera

Hollow viscera

The fracture is the traumatic injury with a break in the continuity of the bone. Its study corresponds to another subject. It is enough to point out here its production mechanism, which can be: by pressure, by twisting and by traction.

The contusion is caused by the impact of a blunt object with the body, through pressure or traction. Ecchymoses (" purples") and hematomas ("bumps") appear.

The wound is the traumatic injury with continuity solution of the skin. They are produced by cuts or traction. They tend to bleed due to the frequency with which the blood vessels are cut. The wounds are called blunt when they are associated with contusion and their edges are irregular, and incised when their edges are regular and smooth.

The puncture or perforating wound is punctate or wider, but extends in depth and frequently affects internal organs.

Internal organs can be injured in the course of open trauma (with a wound that communicates to the outside) and closed trauma (without it). When a solid viscus (such as the liver, spleen, or kidney) is injured, intraparenchymal hemorrhage (hematoma) occurs.

which can break through to the surface of the organ and give rise to hemoperitoneum or a subcapular hematoma.

Injury to a hollow viscus (such as the stomach, intestinal loops or gallbladder) produces a leak of its contents into the peritoneum, which causes a serious inflammatory process called peritonitis.

The most frequent cause of mechanical violence is accidents. In our country they represent the fifth cause of death and include traffic, work and home accidents.

Other types of mechanical violence are suicides and self-inflicted injuries, as well as other injuries and violent deaths in the course of natural disasters, acts of war, homicides and assaults.

Immobility and sedentary lifestyle

The defect or restriction of movement also harms the health of the population. It affects the capacity for physical work, favors obesity, the appearance of diabetes mellitus and coronary, brain and lower limb vascular diseases.

rivers.

PHYSICAL FACTORS

Physical factors can produce pathological disorders when they exceed the adaptability of the subject.

We can group them as follows:

am io temperature

¡Increase: heat

Decrease: cold

am io of pre i atmosphere

rich

2Increase

adiacio e

eletricidad

Heat disorders

High temperatures can produce local and general effects, such as burns in the first case, and heat stroke and syncope in the second.

Thermal burns can be caused by direct flames, boiling liquids, vapors, sunlight, hot objects, and hot surfaces. They are also produced by electric current (electrical burn) and corrosive chemicals (chemical burn).

Thermal injury consists of necrosis due to direct coagulation of the tissues. It is associated with vascular changes with vasodilation and, sometimes, plasma extravasation. Intravascular coagulation may occur. Hypovolemia, infection and shock are common complications in extensive burns. The extent of the burns is important

prognosis: the greater the body surface area affected, the less chance of survival.

Depending on the depth of the injury, burns are classified as epidermal, dermal and hypodermal, according to the skin strata involved.

Heat stroke is an episode characterized by loss of consciousness associated with hyperthermia, in people exposed to high temperature environments, such as boilers, machine rooms, sports or military training in very hot climates. The high ambient temperature prevents the organism from releasing endogenous heat. The skin is hot and dry; the pulse, full and tachycardic.

Heat syncope also associates loss of consciousness and hyperthermia. However, the skin is cold and sweaty, the pulse is weak, and blood pressure is low. This episode is common in large public gatherings with hot environments. The pathogenic mechanism appears to be a vaso-vagal reaction with hypotension and lipothymia.

Disorders caused by cold

Cold can also produce local and general effects. Locally, freezing temperatures can cause coagulation necrosis with burns of the skin and mucous membranes. Epidermis (erythema a frigore), dermis and hypodermis may be of interest. There are progressive vascular and metabolic changes such as vasoconstriction, which may be followed by vasodilation with fluid extravasation and, finally, by intravascular coagulation. Refrigerator gangrene has been described; A frequent form in wars is the trench foot or immersion foot. Metabolic changes operate at the cellular level: metabolic slowing and subsequent suppression, crystallization of intracellular water. Hypothermia is the general effect of cold on the body, common in countries with cold and temperate climates.

Disorders caused by changes in atmospheric pressure

At sea level the atmospheric pressure is 760 mm Hg (1 at) and the partial pressure of oxygen is 160 mm Hg. Underwater, a pressure equivalent to l at is supported for every 10 m of depth descended.

Both increases and decreases in atmospheric pressure can cause disorders. Among the disorders produced by the increase in atmospheric pressure we consider the effects of explosions. These cause a compression expansive wave or positive wave followed by a suction wave or negative wave, which cause serious traumatic injuries to the soft tissues, bones and internal organs. This disorder is known in Anglo-Saxon literature by the name of blast injury.

The decrease in atmospheric pressure causes disease in two different circumstances: due to a sudden change from a normal atmospheric pressure to a low pressure and from a high-pressure environment (deep sea or hyperbaric chambers) to a normal atmospheric pressure.

Ascension to high altitudes involves adaptation to lower atmospheric pressure. Changes in volume and pressure of the gases contained in the natural cavities (perinasal sinuses, middle ear, respiratory and digestive tracts) occur that tend to balance the gradient produced. Likewise, the decrease in the partial pressure of oxygen determines the unsaturation of arterial blood and hypoxia. Depending on time, hypoxia can give rise to polyglobulia stimulated by renal erythropoietin. Acute and chronic mountain sickness has been described in visitors or inhabitants of highlands, cities or regions between 2 000 and 4 500 m or more.

Diver's disease is caused by the formation of nitrogen bubbles in the extracellular fluid as a result of a sudden ascent to the surface from great depths. This does not allow the gradual release into the blood and lungs of the nitrogen previously dissolved in the body fluids, caused by descent into a high-pressure environment. The nitrogen bubbles converge in the blood and become gas emboli that cause serious disorders depending on the vascular region they occlude.

Disorders caused by radiation

Radiant energy includes two different forms of energy propagation: corpuscular radiation, made up of fast-moving particles, and electromagnetic radiation, made up of massless waves of wave motion (Table 22.1).

Chart 1

adiaci	Guy	ecto
Corpuscular	Alpha particles Beta particles	lonizing
Electromagnetic	Gamma Rays Rays	lonizing
	Infrared rays	Non-
	Ultraviolet rays	ionizing

Corpuscular radiation is represented by alpha, beta particles, protons and neutrons, which are produced by the spontaneous or provoked disintegration of radioactive elements.

Electromagnetic radiation covers a wide spectrum of wave motion: electric waves,

radio, infrared rays, visible rays of light, ultraviolet rays, X-rays and gamma rays. Due to their pathogenic interest we will only consider the last two here.

To understand the nature of the different types of radiation, a brief reminder of the structure of the atom is necessary. This consists of a nucleus and its orbit.

The nucleus is composed of protons (positive charge and mass l) and neutrons (no charge and mass 1). The orbit contains the electrons (negative charge and negligible mass), which rotate around the nucleus, imitating the solar planetary system. The sum of protons and neutrons, which are the elements with mass, determines the atomic weight. The number of electrons that, to maintain electroneutrality, is equal to the number of protons, determines the atomic number of the element.

Alpha particles are made up of two protons and two neutrons, just like a helium atom without orbital electrons (charge plus 2 and weight 4). Due to their large mass, they have poor skin penetrability, but not when they are administered orally or parenterally. Due to their double positive charge, they attract orbital electrons from the tissues, causing considerable ionization when released.

Beta particles are made up of elements with a negative charge and negligible mass. They also have little skin penetration. If introduced internally, they exert an ionizing effect by repelling and releasing orbital electrons.

X-rays and gamma rays act on tissues in a similar way. In practical terms they only differ by their origin.

X-rays are produced artificially by bombarding a metallic target by fast electrons, of orbital origin. They have mass and charge equal to zero.

Gamma rays are derived from the nuclear decay of radioactive elements. Like X-rays, they have great ionization penetration.

The pathogenic mechanisms involved in the biological effects of radiation are complex. The most important seems to be the ionization of intracellular chemical compounds. This can lead to:

1. Lethal or sublethal cellular enzymatic damage.
2. Inhibition of DNA synthesis.
3. Arrest of mitosis and mutations.
4. Thrombosis with vascular obliteration.

All cells can be affected and destroyed by ionizing radiation depending on the dose and exposure time. The most sensitive cells are those that are poorly differentiated and poorly specialized, as well as dividing cells. Radiosensitive structures include the hemnolymphopoietic system, the germinal epithelium (gonads), the skin and mucous membranes, and the eyes. As little radiosensitive, the muscular system the skeletal system, the nervous system, the endocrine system and some organs (lung, liver, kidney).

Disorders caused by electrical current

Electricity, both natural (lightning) and industrial, can cause injury and death through two mechanisms:

Electrical burn at the entry and exit points due to the increase in temperature in relation to the skin's resistance to electrical conduction, which determines its transformation into heat energy.

Interruption of nerve conduction due to intracellular ionization, which in turn causes respiratory arrest, ventricular fibrillation and cardiac arrest, especially if vital centers (medulla oblongata, heart) are involved in its path; It depends on the intensity and duration of the current.

UIMAL AGENTS

Different chemical elements present in the environment can cause serious disorders that affect human health. Due to their importance, we are going to refer to the following:

- Intoxication and adverse reactions to drugs.
- Alcoholism.
- Smoking.
-Air pollution.

Intoxication and adverse drug reactions

Toxins can cause local and general injuries depending on their chemical nature and mechanism of action. From a pathogenic point of view, they interfere with intracellular enzymatic mechanisms and give rise to serious metabolic disturbances that may become irreversible (Table 22.2).

Local lesions are related to the area of contact of the agent with the skin or mucous membranes. Corrosive agents (acids and alkalis) cause coagulation necrosis and give rise to chemical burns.

Injuries or general alterations occur at a distance from the area of contact or ingestion. They are variable depending on the chemical nature of the venom and tend to predominate in certain organs or systems.

Alcoholism

The consumption of alcoholic beverages is part of contemporary man and woman. It has been postulated that taking up to one ounce (30 mL) of ethanol daily, contained in 60 mL of rum, whiskey, vodka or similar strong drink, or in 300 mL of wine, or in 720 mL of beer, is beneficial for health mainly due to its beneficial cardiovascular effects. The acute and chronic effects of ingestion constitute a serious health problem at the biological, psychological and social levels.

Chart

structure	Age you	eacci
Nervous system	Ipnotics and sedatives	Depression, coma
	Phenothiazines	Dystonic and parkinsonian syndromes
	Antipsychotics	
	carbon monoxide	Cerebral hypoxia due to excess carboxyhemoglobin that displaces normal hemoglobin
	organophosphates	Excess acetylcholine due to inhibition of cholineste-
		rasa
	methyl alcohol	Destruction of retinal neurons with blindness Medullary
hemolymphopoietic system	Chloramphenicol, butacyfone, sulfonamides, cytostatics	aplasia Agranulocytosis Thrombocytopenia emolysis
liver	Phosphorus, carbon tetrachloride	massive epathonecrosis
	Isoniazid, halothane, paracetamol	diffuse epathonecrosis
	Tetracycline	epathosteatosis
	oral contraceptives, chlorpromazine	Cholestasis
Kidney	Mercury, aminoglycosides	Acute tubular necrosis
	Phenacetins and others pain relievers	Papillary necrosis
Heart	Theophylline	Arrhythmias
	Terfenadine + macrolides	
	Antiarrhythmics	
	Doxorubicin	cardiomyopathy
Lung	Salicylates	Asthma
	Nitrofurantoin	Diffuse alveolar lesion Interstitial
	Busulfan	fibrosis
Bowels	Broad spectrum antibiotics	Superinfection by C. difficile with pseudomembranous enterocolitis

From a medical-legal point of view, alcohol poisoning is considered to exist when an alcohol level of 100 mg/dL is found in the blood. When this level doubles, an alcoholic narcosis can appear and when it triples, an alcoholic coma. The ingestion of alcoholic beverages is involved in a high proportion of accidents, especially traffic accidents with their well-known consequences of death, disability and economic losses. Thus, the love of alcohol has serious consequences for addicts. The alcoholic suffers a progressive deterioration of his personality and becomes involved in serious family, work and social difficulties that can lead to generalized isolation and rejection.

The main consequences of chronic alcoholism on health appear in table 22.3.

i	Tra tor or
Digestive	Acute alcoholic hepatitis. Liver steatosis and cirrhosis
	Pancreatitis, gastritis, oral, esophageal and liver cancer

Chart

i theme	Tra tor or
Highly strung	Polyneuritis. Mernicke-Korsakoff syndrome
	Cerebellar degeneration
	Brain degeneration
Cardiovascular	High blood pressure
	Cardiomyopathy
Musculoskeletal	Rhabdomyolysis
	myopathies
Reproductive	testicular atrophy
	Loss of libido and sexual potency, delay in fetal development and malformations during pregnancy

Taba uism

The habit of smoking, especially cigarettes, represents the most widespread and harmful chemical agent that produces diseases in modern life. In recent years, numerous clinical and epidemiological studies have highlighted the close relationship between the habit of smoking cigarettes and the appearance of different diseases that are becoming more frequent every day (Table 22.4).

Table .4

Respiratory disorders	Lung cancer
	Chronic bronchitis
	Pulmonary
Cardiovascular disorders	Ischemic heart disease
	Cerebrovascular disease Peripheral vascular disease
Digestive disorders	gastroduodenal ulcer
	Gastritis
Disorders genitourinary	Propensity for abortion and premature birth
	bladder cancer
Other neoplasms	Mouth
	Larynx
	Esophagus
	Pancreas

The pathogenic mechanisms activated by cigarette consumption are the subject of intense research. Although much remains to be clarified, different harmful factors contributed by cigarette smoke have been identified:

Carcinogens such as benzopyrene, which promote local or distant malignant cellular changes depending on the dose, exposure time and individual susceptibility.

Irritants, which promote cough, bronchoconstriction, secretion of mucus and interference with normal ciliary self-cleaning mechanisms.

Nicotine, which stimulates the release of catecholamines by the adrenal medulla, which promote an increase in platelet adhesiveness, lipemia and cardiac output, favoring cardiac arrhythmias.

Carbon monoxide, its high content in inhaled smoke determines an increase in the concentration of circulating carboxyhemoglobin and a decrease in oxygen delivery. This hypoxia also contributes to atherogenesis.

Air pollution

Along with oxygen and other natural atmospheric gases that we breathe, countless amounts of polluting elements reach the bronchopulmonary intimacy daily in the form of other gases, fibers, particles and bacteria, which can cause disease, depending on the agent inhaled, its levels in the environment, the size of the particle and the duration of exposure.

The lesions they cause are varied: irritation and inflammation of the respiratory mucous membranes and parenchyma, bronchospasm and hypersensitivity, production of pulmonary emphysema, pulmonary fibrosis and cancer at any level of the respiratory tract.

Different pathogenic mechanisms seem to operate in the determination of fibrosing lesions, including that caused by macrophages. These phagocytose the particles deposited in the pulmonary alveolus and transport them to the interstitium. When macrophages die, particles and enzymes are released, which give rise to an inflammatory and fibrotic reaction. The particles can be phagocytosed again and the phenomenon repeat itself.

More than thirty different forms of pneumoconiosis have been described. The most important are: anthracosis (produced by coal), silicosis (produced by sand or silica) and asbestosis (produced by a complex silicate, asbestos).

BIOLOGICAL AGENTS

In medical pathology, biological agents are understood as those living organisms capable of producing diseases in humans.

Classification

These agents are:

- Fungus.
- Bacteria.
- Rickettsias.
- Viruses.
- Animal parasites.

Fungus. They are multicellular thallophyte plants that do not contain chlorophyll, for example, Histoplasma capsulatum, which causes histoplasmosis.

Bacteria. They are microscopic unicellular organisms of approximately almy size whose reproduction is generally carried out by binary fission, for example, the pneumococcus, the agent of lobar

Rickettsias. They are microscopic unicellular organisms whose size is less than 0.5 m; They differ from bacteria in that they require the presence of living cells to multiply, although their metabolism is independent of the cells present. An example is Rickettsia mooseri, which causes murine typhus.

Virus. They are submicroscopic entities that reproduce only within submicroscopic cells. Its size ranges between 7 mm and 0.5 m. Hepatitis virus A can be cited as an example.

animal parasites. They can be classified into protozoans such as Amoeba histolytica and metazoans such as Ascaris lumbricoides.

Types of dependency relationship between living organisms

The intimate proximity in which the different organisms are found gives rise to different types of relationships:

Commensalism. In which one of the organisms benefits from the products of the other without causing harm.

Symbiosis. Where from the union of two organisms, both obtain benefit, as occurs in lichens, which are nothing more than the symbiotic union of an algae with a fungus.

Parasitism. In which relationship one of the organisms benefits to the detriment of the other.

The organisms of the first two types -commensalism and symbiosis- are grouped under the name of saprophytic agents and those of the last one -parasitism- refer to to pathogenic agents, capable of causing disease.

The dividing line in this relationship is not always clear; Escherichia coli is a saprophytic germ in the human intestine that helps maintain normal flora; However, if it lodges in the urinary tract it is pathogenic, because it causes disease.

Virulence and susceptibility

Virulence. It is the degree of damage that a pathogenic biological agent is capable of causing and depends on its number, and its ability to multiply, spread and produce toxins.

Susceptibility. It is the host's ability to resist the action of a pathogenic biological agent and is related to genetic, ethnic, social, nutritional and health factors.

Aspects to consider in diseases due to biological agents

Fountain. The source of infection is understood to be the reservoir of the pathogenic agents.

The source may be the sick man, as in influenza, or the healthy man (healthy carrier), as has been observed in some typhoid epidemics. It can also be animals or their products, as occurs in brucellosis, or the earth as in tetanus.

Contagion. Contagion is the mechanism by which a biological agent reaches the source of infection to the subject who suffers from it.

It can be congenital, when the infection has occurred in intrauterine life (congenital syphilis). Contagion can also be direct, as occurs in venereal diseases, or indirect, when there is some intermediate element between the source or reservoir and the host. Among these elements we must consider air, water, food, insect vectors and inoculation.

Influenza is transmitted through sneezing and coughing, which, by eliminating multiple microdroplets into the air, reach the recipient's respiratory tract. Typhoid fever is usually transmitted through water;
Dengue, on the other hand, requires the presence of an insect vector. Finally, hepatitis B is contracted by inoculation by transfusing blood containing the virus.

Infection. It is the penetration and establishment of the biological agent in the organism. The site of penetration can be an epithelium: the skin or the mucous membranes.

Invasion. Ability to spread the biological agent or its toxins in the body. As an example of the first type we have meningococcal meningitis, which does not

It is more than the result of meningococcus invasion of the upper respiratory tract to the meninges.
Corynebacterium diphteriae, the germ that causes diphtheria, is an example of the dissemination of toxins that tend to settle in the myocardium and nervous system.

Mechanisms of action of biological agents to cause disease

Five mechanisms have been invoked:

1. Multiplication and propagation of the agent. Example the pneumococcus.
2. Toxin production. Clostridium tetani produces a neurotropic exotoxin that causes tetanus.
3. Multiplication and propagation of the agent linked to toxin production. It is observed in diseases caused by Staphylococcus aureus.
4. Mechanical effects. A group of Ascaris lumbricoides can clump together and lead to intestinal obstruction. nal.
5. Hypersensitivity. Anaphylaxis may be observed due to the rupture of a hydatid cyst.

Organic defense mechanisms against the action of biological agents

Against penetration

The integrity of the epithelia, epithelial desquamation and mucous secretions prevent entry and drag the germs with them. Coughing, sneezing, vomiting and diarrhea are mechanisms for the sudden expulsion of pathogenic agents that make their penetration difficult. The acidic pH of sweat and gastric juice tends to neutralize pathogenic germs.

Against multiplication and

invasion Inflammation. The organism tries to encompass the germs in a fibrin network, causing their phagocytosis by polymorphonuclear leukocytes in the phenomenon known as inflammation; Later, in chronic inflammation, phagocytosis is usually carried out by mononuclear cells.

Antibody production. Biological agents, through their protein components or polysaccharides, are capable of generating the production of antibodies that tend to neutralize them or their toxins.

properdin system. It has been described that the combined action of a plasma globulin, the C 1 and C4 fractions of the complement and the magnesium ion, form a kind of primitive antibody, capable of combating biological agents, which has been called the properdin system (from the Latin, pro: in favor; will lose: to destroy).

Interference. It is the ability of cells that have been affected by a virus to resist it upon new exposure, without the mediation of immunological mechanisms of antibody production.

Diagnosis of diseases due to biological agents

A medical history should be taken with a careful history and a complete physical examination. In epidemics it is necessary to take into account the epidemic genius.

The diagnosis of certainty is established by direct demonstration of the agent or proof of its presence by indirect means.

Among the means of direct demonstration we have simple staining and extension, cultures, inoculation to animals, demonstration of the agent in a biopsy and in cases of spirochetes, dark field study.

The indirect means that we can mention are: demonstration of antibodies, serological tests, skin tests and presence of typical lesions such as rectal ulcers of amebiasis.

GENETIC FACTORS

When studying genetic factors as the cause of a large number of diseases, we must keep in mind that every character results from the interaction of genetic and environmental factors.

In medicine, the fundamental role of genetics is to determine its participation in the etiology of various diseases or defects, delimiting possible environmental influences.

Thus we find disorders in which defects in genetic information are of greater importance, as the main etiology or vice versa, but the combined action of the genome-environment binomial must always be remembered.

To develop genetic disorders in a general way, certain basic concepts and some of the categories used by geneticists must be remembered, such as:

Gen. It is the functional unit that governs inheritance. It is made up of DNA of different lengths that have the necessary information to produce the synthesis of a polypeptide chain that, alone or joined to others, can have enzymatic, structural or transport functions.

This information is transmitted from generation to generation by self-replication of DNA chains and is translated from the language of bases to that of amino acids in proteins, through the mechanism of protein synthesis, for which RNA intervenes in its three known forms: messenger, ribosomal and transfer, through a code of triplets of bases. Each gene occupies

pa a place on the chromosome, which is called a locus.

Chromosomes. They are made up of DNA and proteins and are the result of the coiling of DNA chains during cell division, the only moment in the cell cycle when they can be studied. In men there are a total of 46, of which 44 are autosomal and 2 sexual (XX in females and XY in males).

This number is reduced by half during gametogenesis, leaving 22 autosomes and one X chromosome in the egg, and 22 autosomes and one X or Y chromosome in the sperm.

The classification of chromosomes is based on the size and relative length of their arms. There are 23 pairs and groups, designated A to G, which constitutes the karyotype.

Phenotype. This is what we can see as a result of the genotype. The term phenotype defines the expression of a character determined by a specific gene.

It can be studied at various levels, ranging from the simple observation of the individual through a clinical history, to the analysis of the results of neurophysiological, hematological, radiological, biochemical, immunological studies, etc. The deeper the study of the phenotype, the closer we will get to knowing the genotype.

Genotype. It is the genetic constitution of an individual. The term genotype is used to refer to the dose of the mutated gene in the genome of the individual under study, that is, to the genetic composition for a specific trait.

Sudden and hereditary changes in genetic material are called mutations. The presence of a mutation indicates that there is a non-mutated gene, which expresses a character that is recognized as "normal" or common, without unfavorable repercussions for the individual. This "normal" gene is known as wild type.

The presence of a mutation of the wild type gene determines the existence of two alternatives for that locus (site occupied by the gene on the chromosome). These two alternatives, mutated gene and wild type gene, are defined with the term alleles. These alleles occupy the same locus on homologous chromosomes.

Homozygous, heterozygous and hemizygous. The diploid character of the genome determines that each individual must combine the presence of these alleles, in combinations of two.

The genotype can be homozygous, if both alleles of the two genes are the same; heterozygous, if the genes

They are different and hemizygous when in the genotype there is only one of the representatives of a pair of alleles. This last genotype is typical of the male sex when we refer to genes that are located on the

Dominant and recessive character. The terms dominant and recessive are used to refer to the dose that these alleles require to express themselves in the phenotype. For example, if the mutated gene is expressed in heterozygous individuals, the character it expresses is dominant, but if it requires a homozygous genotype for its expression it will be considered recessive. Sometimes both are expressed equally in a heterozygous state, defined

thus becoming co-dominant.

Mendel's Laws

These laws correlate with meiosis in the process of gametogenesis or production of eggs and sperm.

matozoids.

Mendel's First Law or the segregation of genes.

It states that the genes arranged in pairs are segregated or separated in the gametes, since when the homologous chromosomes separate, the paternal genes are segregated from the maternal ones in the chromosomes. This first law is the result of a monohybrid crossing, that is, of a single character.

Alleles are segregated or separated according to Mendel' s first law and refers to the segregation analysis of a single pair of alleles.

Mendel's Second Law or independent segregation. It states that loci that are very separated or on different chromosomes segregate into gametes independently of each other. This law is the result of dihybrid crosses, that is, crosses of two different characters.

That is, the second law adds that the alleles segregate independently and at random, when it refers to two pairs of alleles that express different characters.

genetic alterations

The human genome is composed of nuclear DNA and mitochondrial DNA. Nuclear DNA is contained in 46 chromosomes that interact harmoniously from the zygote until the end of life. A mutation in a gene can go from unnoticed to compromising the health and phenotypic harmony of the individual to varying degrees of severity.

The effect of a mutation can be expressed as an abnormality that can be detected at birth due to its phenotypic characteristics or at any time.

after birth, compromising not only the quality of life and its reproduction, but even life itself.

Multiple mutations have been described, ranging from a simple molecular substitution in one of the nitrogenous bases that make up DNA to alterations that even compromise the total number of chromosomes or their structure and that can be detected by cytological techniques. There are also mutations that compromise perhaps only one of the genes that act as a group and whose actions add up and are expressed in a specific phenotype.

As the mutations are expressed in some way, they have been classified based on the common aspects that allow us to identify their phenotypic and family characteristics as: monogenic, chromosomal and multifactorial. Therefore, according to the type of mutation, there are also three fundamental types of genetic disorders or defects:

1. Alterations or defects that are the result of single gene or monogenic mutations. Monogenic mutations follow Mendelian patterns of inheritance, which is why they are also called Mendelian inheritance. There is evidence of a progressive increase in their number, which now reaches just over 9,000 entities.

2. Alterations or defects as a result of chromosomal disorders. In this second type there are potentially as many defects as there are chromosomes and their increase corresponds to the technological advances that allow their detection. Its incidence is around one in every 160 births.

3. Multifactorial alterations or defects. In this type there is a high number of malformations that are evident in approximately 4% of newborns, in addition to other alterations that appear in adults, which constitute so-called common diseases of which around 60% present a multifactorial genetic defect.

Alterations or defects that are the result of single gene or monogenic mutations

We must define other terms, in addition to those already seen, that are used in the analysis of family segregation of monogenic mutations and that are related to the phenotypic expression of the mutation:

Penetrance is the term used to refer to the expression in terms of all or nothing. If the mutation is expressed in less than 100% of the carrier or heterozygous individuals, it is said that the mutation has a reduced penetrance and that individual who is apparently "healthy for the character or disease being studied in the family can transmit the mutation to his offspring and they express the defect. The reduced penetrance appears be the effect of the relationship of the mutation in question and other genes in the genome with which it is interacting.

Expressivity is used to refer to the degree of severity that manifests in the phenotype in clinical terms, it is synonymous with severity. The expression of a gene also depends on its relationship with the rest of the genome, but also on the genome-environment relationship.
To refer to these phenotypic gradations, the term variable expressivity of the gene or mutation is used.

Pleiotropy or pleiotropic effect of a gene is the reference to all the phenotypic manifestations in different organs or systems that are explainable by a simple mutation. A classic example to explain this term is Marfan syndrome, whose mutation affects the FBN1 gene that encodes the protein fibrillin. This protein is found in the connective tissue and explains the skeletal, ocular and cardiovascular manifestations that characterize the syndrome.

Monogenic nuclear defects determine the existence of four types of inheritance according to the location of the mutated gene on autosomal chromosomes or on the X chromosome and according to the mutated gene dose necessary for the expression of the mutation in the phenotype.

These inheritances are:

- Autosomal dominant.
- Autosomal recessive.
- Dominant linked to the X chromosome.
-X-linked recessive.

Each one can be recognized in the analysis of the family tree, an important instrument for its determination.

The recognition of these four types of inheritance is based on the analysis of family segregation, specifying:
who is affected, what sex they have and what family relationship unites them.

Autosomal dominant inheritance

In this type of inheritance, the character is expressed in 50% of the offspring of the affected person, which is generally heterozygous, because the appearance of homozygous patients is unlikely, since marriages between both affected individuals are very rare. The affected person has a 50% chance of transmitting the mutated allele to his or her offspring; the character appears in each generation of the affected family; Healthy individuals do not transmit the character to their children and since it is an autosomal inheritance, both females and males can be affected.

An example of this appears in Figure 22.2.

We can symbolize the mutated gene with the capital letter A and the wild type with the lowercase letter a. The genotype

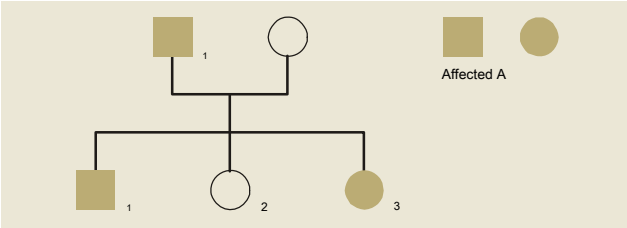


Fig. 22.2 autosomal dominant erence.

of affected individuals will be heterozygous (Aa) and that of unaffected individuals will be homozygous recessive (aa).

Pairs I-1 and I-2 will be genotypically A and aa, respectively, and 50% of the gametes of individual -I will carry the A mutation or the wild-type gene a, while 100% of the gametes of I-2 will always carry the gene

This means that the affected individual who is heterozygous Aa will transmit the defect (mutation A) to 50% of his descendants, regardless of the sex of his children. In this type of inheritance, the absence of other family members affected by the same disease and the absence of maternal or paternal history of the disease, in an individual affected by mutation A, is explained by the appearance of a de novo mutation in the family in question, starting from the first affected individual. At least 8,005 mutations with dominant expression of genes located on autosomal chromosomes have been reported. An example of this type of inheritance is seen in Marfan syndrome.

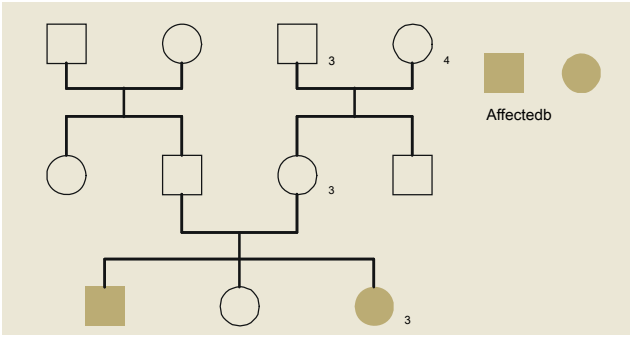
Autosomal recessive inheritance

Here the patient's parents are normal, but obligate heterozygotes, since the patient receives the affected gene from both parents; Let us remember that the recessive character is expressed only in the homozygous. Generally, 25% of the patient's siblings may suffer from the disease, both males and females are affected equally and it is frequently found in consanguineous marriages.

An example of this type of inheritance appears in Figure 22.3.

The parents of affected siblings are healthy or unaffected, like the rest of the family. Here you can-

Fig. 22.3 autosomal recessive erence.



If we designate the mutated gene with the lowercase letter b, normal individuals will be genotypically homozygous (BB) or heterozygous (Bb).

The couple II-2, I-3 will be genotypically heterozygous (Bb) while their children will be genotypically homozygous recessive (bb), in this case when the existence of the two mutated genes is required for the defect to be expressed, the inheritance is autosomal recessive. The heterozygous couple will have a 25% probability of having another sick child regardless of their sex. Table 22.5 illustrates this situation.

Chart

amethyst		
b	BP	Bb
b	Bb	bb

1730 mutations have been reported, with recessive phenotypic expression, of genes located in any of the 22 pairs of autosomal chromosomes.

An example of this type of inheritance is observed in sickle cell anemia, the most common Mendelian genetic disease in Cuba.

X-linked dominant inheritance

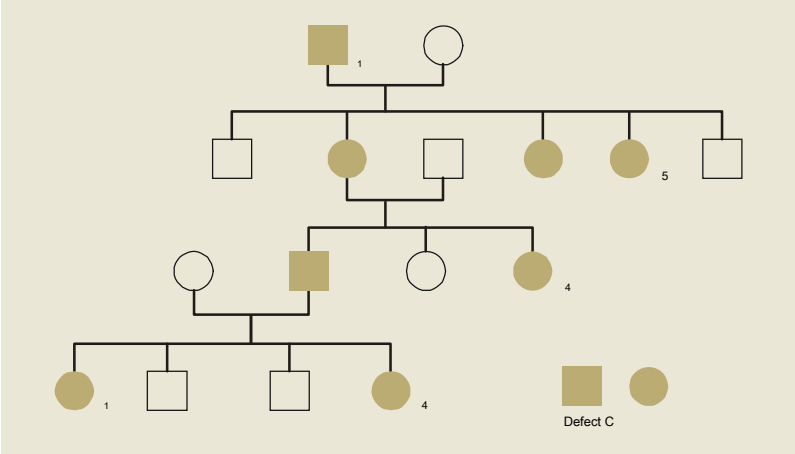
The affected man transmits the disease to 100% of his female daughters, since they will always receive the X chromosome from the affected father, and since it is a dominant character, the heterozygous female expresses the disease. However, through family studies it is difficult to know and identify this type of inheritance and to differentiate it from autosomal dominant inheritance it is necessary to carefully observe the offspring of the affected male, who transmits the disease, as we have already said, to all his female daughters, but never to his male sons. An example of a disease that follows this pattern of inheritance is vitamin D-resistant rickets, hypophosphatemia. An example of this appears in figure 22.4.

If we carefully observe the family tree, it should draw our attention that the defect is due to a mutation with dominant expression, since it is true that affected individuals have one of their parents also affected. However, let us note that in this example, sick men never transmit the disease to their sons, while they transmit the X chromosome to all their daughters and with it, the C mutation, so they will all express the disease, like their father.

In this case, the mutated gene C is located on the X chromosome, and like the X and Y chromosomes

Fig. 22.4 dominant erence linked to

chromosome



are involved in sex determination, individuals with the affected genotype must be analyzed based on their chromosomal sex, that is, XX 1 for women and XY for men. As the mutated C gene is located on the X chromosome, women may have three genotypes: XC/XC, XC/Xc and In man, only two genotypes are possible: XC/Y and Xc/Y, in its homozygous nature. The healthy man in this example will have an Xc/Y genotype. We assume that the genotype of I-l is hemizygous XC/Y and I-2 is homozygous recessive unaffected Xc/Xc. The gametes of I-1 (sperm) will be of two types based on the presence of the sex chromosomes: XC and Y. Note that the mutated C gene is transmitted with the X chromosome.

The gametes of I-2 will all be Xc. This couple will have a probability of having affected offspring depending on sex. While all his daughters will phenotypically suffer from the disease and will be genotypically heterozygous, the sons of this affected man (I-1) will always be genotypically and phenotypically unaffected.

The affected II-2 woman, being heterozygous, will transmit her

X-linked recessive inheritance

Figure 22.5 shows an example of recessive inheritance linked to the X chromosome. In this case the mutation is recessive and differs from the previous inheritance because the daughters of the affected hemizygous man (genotype

Here it is highlighted that it is usually men who are affected and that they are related to each other through heterozygous women.

At least 495 mutations of genes located on the X chromosome have been reported. An example of a genetic defect with this type of inheritance is observed in hemophilias A and B.

If we mark with the letter Hel the normal gene and the allele that gives rise to hemophilia (a classic example of this type of he-

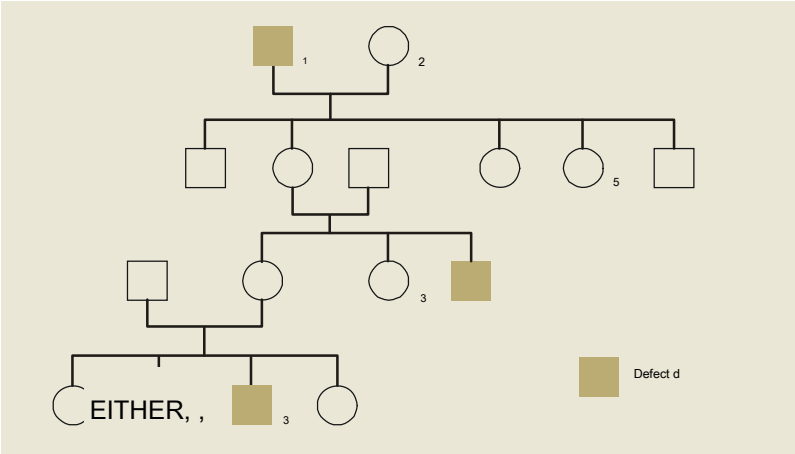


Fig. 22.5 recessive erence linked to

chromosomesSoma

rence), the female genotype can present in three forms: XHXH; XHXh and XXh; while the masculine will be XHY or XY. When analyzing the behavior of recessive diseases linked to the X chromosome in a family, we will observe that there are more males affected; The character is transmitted from the affected father to the daughter and from this to half of his male children, who will present the disease, while half of his female daughters will be heterozygous, who are called carriers, and the affected father never transmits the disease directly to his male children; Another example of this type of inheritance is pseudohypertrophic or Duchenne muscular dystrophy.

There are also monogenic defects that determine the existence of two types of non-Mendelian inheritance, which are:

- Inheritance linked to the Y chromosome.
- Mitochondrial inheritance.

Y-linked inheritance

The Y chromosome has a segment at the end of the short arms called the pseudoautosomal region (through this region the X and Y chromosomes remain joined together during cell divisions). The segregation of the genes located in this segment is not different from autosomal inheritance, but the genes located in the rest of the Y chromosome are segregated only through males who present the mutation. At least 27 mutated genes have been reported on the Y chromosome. Examples are Y-linked retinitis pigmentosa, azoospermia factor 1, gonadoblastoma, histocompatibility antigen Y (HY), interleukin-3 receptor, testicular determining factor (TDF), sex determining region Y (SRY).

Mitochondrial inheritance

There are other monogenic mutations that differ from the previous ones because the affected or mutated gene is in the mitochondrial DNA. Mitochondria have independent intracellular reproduction (by fission) and although both females and males are affected, affected men do not transmit the disease since the sperm does not contribute mitochondria to fertilization. Mitochondria are only transmitted through the egg, whose cytoplasm is much larger.

An example of mitochondrial inheritance is shown in Figure 22.6. It is difficult to make a predictive analysis of the probability that an affected woman will have healthy or sick children, since this depends on the number of normal and abnormal mitochondria in each egg.

60 mitochondrial mutations have been reported. Examples of this type of mutation are those observed in

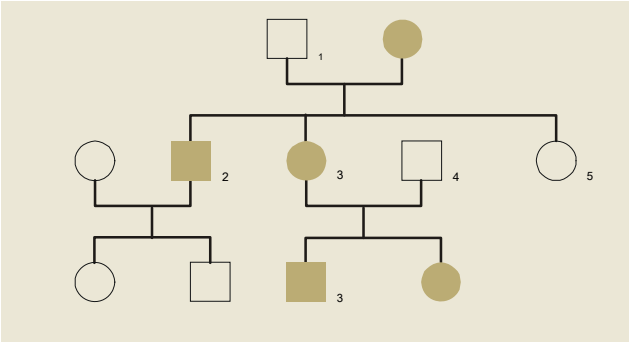


Fig. 22.6 Mitochondrial erence.

chloramphenicol-induced anemia, Lebery optic atrophy and diabetes-deafness syndrome.

Alterations or defects resulting from chromosomal disorders

Current technology has made surprising advances in the study of human chromosomes. Cytogenetics by itself or expanded with molecular techniques allows the detection of increasingly smaller defects that reduce the gap between monogenic and chromosomal mutations. These defects are called chromosomal aberrations (CA), and may have their origin in alterations in the number (aneuploidies or polyploidies) or in the structure of the chromosome.

Chromosome.

Aneuploidies are due to a failure in the segregation of chromosomes into gametes during meiotic division. It is in this process where the number of chromosomes in the germ cell (diploid) is reduced by half (haploid), so that during fertilization the diploid number characteristic of the species is restored.

Each gamete must have 23 chromnosomes, but if a gamete with 22 or more than 23 chromosomes participates in fertilization with a normal gamete, a zygote with 45, 47 or more chromosomes will be created. This imbalance will be expressed in phenotypes ranging from reproductive failure to mental retardation. Of course, there are intermediate situations consisting of multiple malformation syndromes with gradations in severity that will be related not only to the defect or excess of DNA, but also to the chromosome involved.

Aneuploidies, in turn, can occur in both autosomal and sex chromosomes. In autosomal individuals, the most common example is Down syndrome or trisomy 21; Other known examples are trisomy 18 and trisomy D, although new syndromes appear every day due to aneuploidies of other

chromosomes.

Aneuploidies of the sex chromosomes include Turner syndrome (45,XO),

Klinefelter (47,XXY) and other less frequent ones such as tetra X, penta X, etc.

Structural chromosomal aberrations are characterized because there are always DNA breakpoints that determine rearrangements important enough to be observed by cytogenetic techniques. These mutations can be balanced, that is, a phenotypically normal individual who presents chromosomal abnormalities whose clinical manifestation is practically limited to reproductive failures (abortions or children with multiple phenotypic defects sometimes incompatible with life) who have inherited from the father, with the balanced chromosomal aberration, some of the chromosomes affected by the rearrangement of the genetic material. The most important structural chromosomal aberrations are unbalanced, that is, the affected individual always expresses in his or her phenotype some abnormality whose severity depends on the chromosome involved and the magnitude of the defect.

Structural chromosomal aberrations are characterized by:

– Loss of a segment of the chromosome or deletion.

- Duplicated chromosome segments or duplication.

- Abnormalities in the separation of chromaids or isochromosomes.

- Exchange of DNA between two chromosomes or translocations.

Breakage and inverted repair of a chromosome segment or inversion.

The first three are of the unbalanced type and the last two are generally presented as balanced.

The phenotypic expression of these defects when the karyotype abnormality is unbalanced depends, as we have already mentioned, on the type of defect, the magnitude of the mutation and the chromosome involved. For example, when it comes to autosomal chromosomes, the most frequent and notable clinical manifestation is mental retardation, but, in addition, there may be visual, hearing or motor disabilities. When sex chromosomes are involved, the phenotype correlates with the number of X or Y chromosomes involved and with the magnitude of the structural defect. Generally these defects cause variable disabilities that include everything from learning disorders to mental retardation or behavioral defects.

Phenotypic defects in people with balanced chronosomal aberrations are generally expressed in the reproductive stage, presenting reproductive failures ranging from infertility and spontaneous abortions to multiple malformations and neonatal death.

An example of structural aberrations due to deletions or loss of a fragment of a chromosome is the deletion of the short arm of chromosome 5 or cat cry syndrome, due to the supposed similarity of the child's cry with the cat's meow.

Isochromosone, a defect in the separation of sister chromatids, gives rise to chromosomes with the same (iso) genetic information on both arms, causing partial monosomies or trisomies. When this occurs on the X chromosome, phenotypes similar to Turner syndrome occur.

The translocation or transfer of genetic material between non-homologous chromosomes can give rise to Down syndrome due to Robertsonian translocation or centric fusion between the chromosomes of the D and G groups. In this case, one of the parents may be an asymptomatic carrier of the translocation and the probability of having a child with Down syndrome is considered a high risk of recurrence.

Multifactorial alterations or defects

These genetic defects or mutations involve one or several of the genes that together express a character;
They are also known as multifactorial inheritance. There are groups of genes or polygenes in the human genome that participate simultaneously in the expression of traits and that, therefore, are subject to, or are more susceptible to, modifications determined by environmental factors. These multifactorial defects are the cause of the variations that appear in population studies of characteristics such as intelligence, height, blood pressure values, that is, quantitative characteristics. Sometimes several individuals with similar defects appear in a family without being able to determine criteria to define Mendelian inheritance. This is because in specific families there is a greater probability that several members have more similar genotypes, and are more susceptible to environmental variations. To define situations like this, the term family segregation is used.

Other polygenes act in morphogenesis and in cases of mutations the genotype may be more susceptible to maternal environmental defects that could also be nutritional. In these cases the expression consists of isolated congenital defects with variable gradations in terms of aesthetic or functional severity or both for the individual. Examples of these defects are neural tube malformations such as encephaloceles, meningoceles, skulls, facial defects such as cleft lip with or without cleft palate. Diseases such as epilepsy, schizophrenia, ischemic heart disease, bronchial asthma, whose genetic alteration is not due to a simple basic defect and various

Mechanisms determined by several genes are called, within this genetic etiology, complex diseases.

Genetic categories to take into account in genome variations Impact on the interpretation of segregations of specific mutations

chromosome activation

It has been observed that in female soma cells (46,XX), only one of the two X chromosomes is active. The other remains inactive and appears in interphase cells as a dense, strongly colored body on the periphery of the nucleus that is called the Barr body. The inactivation of the The selection of the X chromosome to be inactivated is a generally random phenomenon; Taking into account that when fertilization occurs, each X chromosome has both maternal and paternal origin, in some cells the maternal X (X) will be inactivated and in others the paternal X (X). Once one of the two X chromosomes is inactivated, the descendant cells will maintain the same inactive X chromosome, creating an active cell clone (X) or (X). That is, at the beginning of inactivation, it is random, but once it occurs, the same X chromosome that was inactivated in the first cell of the clone is maintained. The inactivation of this chromosome determines genetic and clinical consequences:

Dose compensation. It equates the dose of gene products with the hemizygous for genes located on the X chromosome, determining similar protein concentrations in both sexes, for X-linked genes.

Variations in the expression of mutations in heterozygous women. For example, presence of more or less severe symptoms in female carriers of hemophilia A or B, Duchenne muscular dystrophy, and X-linked recessive retinal dystrophies.

The female organs behave like mosaicisms.
This phenomenon is observed in recessive ocular albinism linked to Xo in the immunohistochemical test for the detection of dystrophin in women heterozygous for Duchenne muscular dystrophy.

genomic imprint

Investigations of genetic behavior in different species of sexual reproduction, including humans, have accumulated enough evidence that definitively breaks with the belief that the genome haploid contained in the gametes does not present differences that prevent the Mendelian expression of a mutation. A new genetic category appears under the name of genomic imprinting and is defined as the imprint left in the genome of the new individual by the maternal and paternal haploid chromosomal contribution.

This means that a mutation can have variations in its expression in such a variable range that it can range from no expression to extreme severity, depending on whether the mutation has been inherited from the father or the mother.

Examples of evidence of genomic imprinting in humans are the syndromes produced by deletion of the ql1-q13 region of chromosome 15. Given that this chromosomal aberration is inherited paternally or maternally, the Prader Willi or Angelman syndromes will be expressed respectively, whose phenotypes are very different from each other.

Other examples are related to the increase in the severity of the phenotypic expression of the mutation, as has been proposed in myotonic dystrophy to explain the neonatal form that is observed in children of affected mothers who, in turn, have inherited the mutation via
maternal.

Uniparental disomies

The discovery of uniparental disomies emerges as a result of observations in the use of biotechnology, based on molecular characterizations of various mutations. A chromosomal pair is not always made up of chromosomes transmitted in the male and female gametes. It is not known how many times a chromosome pair is the result of a nondisjunction in either of the two meioses of oogenesis or spermatogenesis, but there is abundant evidence that a specific chromosome pair has been transmitted to the egg or sperm by a single parental route, giving rise to a maternal or paternal uniparental disomy.

The phenotypic impact of this phenomenon is the subject of current study and it can be predicted that its variations will depend on the genes of the chromosomes involved and the type of disomy, which can be isodisomies (defect generated in the second meiosis) or heterodisomies (defect generated in the first meiosis).

Dynamic mutations

This term is applied to genes that present in some region of their structure base triplets repeated in a number of times such that it defines a so-called normal allele or gene. The mutation consists of the increase in the number of times that the triplet is repeated and its expression is corresponding.

weight with this increase. It is a gene that grows and its growth has a limit beyond which its expression can be null and cause a developmental defect.

The syndrome from which this new genetic category is introduced is called fragile X syndrome. The mutation of this syndrome consists of an increase in repeats of the CGG triplet at the 5' end of its first exon, whose normal allele has an average of 30 repeats.

This increase occurs in stages. Hence the term gene that grows. The first growth or premutation ranges between 43 and up to 200 repetitions without having a very specific phenotypic effect. After 200 repetitions, the complete mutation occurs and the syndrome is expressed.

This gene is found in the q27.3 region of the Xy chromosome, therefore, it is inherited linked to the This type of mutation explains the genetic phenomenon of anticipation or observation of the earlier appearance of symptoms in new generations, independent of the sex of the individual who transmitted the mutation. Anticipation is a common phenomenon in progressive genetic diseases of the central nervous system. Some examples are represented, in addition to fragile X syndrome, by myotonic dystrophy, Huntington's chorea, Holguín-type ataxia, among others.

Germinal mosaicisms

These are mutations that appear in germ cells that, in turn, give rise to affected gametes in a number or with a probability that will depend on the number of germ cell generations with the mutation. The suspicion of this type of mutation is the recurrence of a specific defect that appears to be a new mutation with a dominant character and that, without a previous family history, is repeated in two or more children, appearing, when it occurs in the same couple, to be an autosomal recessive inheritance. The most illustrative example is achondroplasia, a disproportionate short stature whose mutation is characterized by a base change in the receptor gene for fibroblast growth factor 3 (FGGFR3). A simple change of one nitrogenous base for another is expressed by the bone dysplasia that characterizes this mutation.

Genetic heterogeneity

This term deserves special attention, as it applies to mutations in genes located in different chromosomes that produce similar expression in the phenotype (non-allelic heterogeneity) and mutations that affect different sites of the same gene (allelic heterogeneity). This category extraordinarily complicates the etiological study of developmental variants of genetic origin and constitutes a broad and fundamental source of developmental genetic diversity.

Digenic inheritance

The expression of a defect is due to the coincidence of a double heterozygous genotype for mutations that in a single dose and in isolation do not express the defect in question. An example is illustrated in double heterozygous cases for a form of retinitis pigmentosa, where the person is only affected by this type of retinitis pigmentosa if he or she is heterozygous for a mutation of the gene "outer segment protein 1" (RON1) found on chromosome 11 and this coexists with the mutation of the gene that encodes the peripherin protein and is found on chromosome 6.

Loss of heterozygosity

It is a characteristic phenomenon of the expression of tumor suppressor genes. It occurs in various types of cancer where there is a first mutation that determines the presence of a heterozygous genotype and a second mutation on the wild type or non-mutated gene determining a homozygous recessive or hemizygous genotype, if this second mutation determines the loss of the chromosome not affected by the mutation, losing the heterozygous genotype and developing the tumor. It is known as "Knudson's two-hit hypothesis." In hereditary retinoblastoma, the first mutation is germline, that is, the individual is heterozygous for the first mutation. The second mutation has an earlier effect and in multiple cells, which explains why this type of malignant tumor develops in these cases in the first year of life, in both eyes and multiple times. Another example in which this phenomenon is observed is in neurofibromatosis 1l (NF1). THE neurofibromas that characterize this genetic disease are the effect of loss of heterozygosity of the gene that expresses the protein neurofibromin, which also has a cell cycle regulation function, resulting in being a tumor suppressor gene.

Effects of prenatal environmental agents as simulators of genetic mutations

Embryo-fetal development consists of a complex process of molecular and cellular differentiation that begins with the fertilization of the egg with the sperm.

This process can be affected by abnormalities of the genome of the types already explained, but it can also suffer alterations due to the abnormal presence of agents.

environmental factors that interfere with genetic mechanisms that are normally occurring rapidly and with great synchronicity. Each moment of embryofetal development presents cells that can potentially be targets of external agents. These agents that interfere with embryo-fetal development will have various expressions corresponding to their origin, the moment of development in which they appear, the dose, the duration of their presence and the genetic susceptibility to the effect of the agent.

Taking into account the moment of embryo-fetal development in which they act and the effect produced by these agents, they can be classified as teratogens, when they appear in the period of organogenesis (weeks 1 and 2), they cause loss or abortion (between weeks 3 to 8), they determine various types of malformations and after week 8 (fetal period) only minor malformations, functional defects or both are detected.

Other agents only affect the period of fetal maturation, causing functional alterations, and others interfere only with nutrition mechanisms, apparently determining only growth defects.

Teratogenic agents, for the most part, have effects of three types, depending on the moment of development in which they appear, and some of them act as mechanical agents in nature, which can interfere with embryo-fetal development, causing deformities or destruction of tissues, subsequent to the embryonic period.

Many times, from a practical point of view, it is difficult to differentiate between the effect of a teratogen and a genetic mutation. The interrogation is fundamental in the diagnostic order. The environmental agents that have these potentialities on development from organogenesis to fetal development are classified, according to their nature, into biological, physical and chemical agents.

Biological agents, in turn, are classified as viral (cytomegalovirus, herpesvirus, varicella-zoster, rubella, influenza, and HIV), bacterial, spirochetes (syphilis, leptospirosis), fungi, protozoa (Toxoplasma gondii).

Among the physical agents are radiation (dose in excess of 25 rad), the doses received from routine radiological studies, even in the abdomen, do not exceed 0.5-2 rad and do not offer risks, although they do cause great alarm in pregnant women; High and sustained hyperthermia can damage the developing central nervous system.

Physical agents could include the effect of mechanical forces that can affect the process of embryo-fetal development by decompensation of the biological mechanisms created to protect the product.

For example, compressions due to malformations of the uterus, fibroids, which can directly affect fetal parts and cause a deformity or compromise placental blood flow or cause alterations in the blood flow of a fetal part causing asymmetric developmental abnormalities. Other mechanical forces are produced by rupture of the membranes that protect the fetus (amnion); This produces fibrous bands that can trap the product at any stage of development, causing everything from severe malformations to disruptive defects of a fetal part. Amniotic fluid loss is also a mechanism of fetal compression.

The largest group of substances that affects embryofetal development is made up of chemical agents and potentially with the introduction of new products used as medical treatment, these could increase. Substances such as anticonvulsants, anticoagulants, folic acid antagonists, vitamin A derivatives, sedatives, and many others such as thalidomide, produce severe malformation syndromes when used in the embryogenesis period, and functional defects when consumed during the fetal period. In this group, the effect of drugs that, such as cigarettes and alcohol, are widely consumed and generally leave their mark should be highlighted.

Daughters of mothers who smoke more than a pack of cigarettes per day weigh 300g less than expected. Children of women who ingest 3 ounces or more of absolute alcohol per day can develop fetal alcohol syndrome that is manifested by weakness, motor coordination defects, hyperactivity, attention and language defects, strabismus, sensorineural and conductive deafness, and malformation defects. A lower alcohol consumption of up to 1 or 2 ounces produces subclinical defects that are only reflected in the school period.

It is important to highlight that imbalances in the maternal metabolic endocrine balance can also affect embryo-fetal development. Maternal diseases such as diabetes mellitus, hyper or hypothyroidism, hyper or hypoparathyroidism, deficiencies of folic acid and other vitamins, rickets, hypocalcemia, phenylketonuria, among many, can be the cause of severe and varied disabilities of prenatal origin.

In summary, there is a significant number of biological, physical and chemical environmental agents that act on development from organogenesis to fetogenesis, responsible for a great variation in the severity of disabilities that simulate genetic mutational defects, only distinguishable following a logical sequence of clinical and paraclinical studies required to elucidate whether many of these disabilities that have evidence at birth are

of genetic origin (monogenic, chromosomal or multifactorial) , environmental (due to biological, physical or chemical agents) or non-specific (when, despite evidence of a prenatal defect, it is not possible to identify the precise etiological factor).

study methods

There are numerous study methods on which the geneticist relies to reach the etiological factor:

1. Detailed analysis of the patient's phenotype.
2. Analysis of the family study through the creation of family trees.
3. Cytogenetic and dermatoglyphic studies (patterns of grooves in the skin of the palms, fingers and toes).

4. Sexual chromatin (by simple scraping of the oral mucosa) and karyotype study (by peripheral blood culture).
5. Biochemical analysis, which includes qualitative reactions to determine metabolic alterations, protein electrophoresis, etc.
6. Physiological and immunological studies.
7. Others that contribute to defining the role of genetics as an etiological factor of the disease under study.

More specific genetic study methods

- Determination of substrates affected by blockages in a metabolic pathway: study methods such as qualitative determinations in urine, determinations of amino acids, oligosaccharides, mucopolysaccharides and lipids by thin layer chromatography and quantitative determinations of amino acids, organic acids, trace elements, etc.

- Determination of the activity of specific enzymes, after clinical and biochemical evaluation: methods of analysis of enzymatic activity in leukocytes, erythrocytes, fibroblast cultures, and others.
- Determinations of phenotypes of proteins of enzymatic type, transport, etc.: electrophoretic methods or isoelectric approach; Examples: types of hemoglobin, alpha 1 antitrypsin phenotypes, transferrin phenotypes, etc.

Studies for the diagnosis of chromosomal aberrations: methods without cell culture for the observation of Barr bodies or Y bodies that allow the nuclear identification of chromatin corresponding to chromosomes of the number ro and the structure of the chromosomes, by classifying them according to their size and shape, as well as the banding pattern used for their longitudinal differentiation. This classification is called karyotype (classification of the 23 pairs of diploid chromosomes). This analysis includes the differentiation of each chromosome pair, by applying G,0, R, NOR, C banding techniques, as required.

High-resolution cytogenetic or prometaphasic studies, for the detection of small structural defects not detected by metaphasic methods.

- Molecular cytogenetic methods using in situ hybridization (FISH), by applying, after denaturation of the chromosomal DNA, complementary and marked DNA segments that facilitate the identification of the chromosomal segments with which it can hybridize taking into account the phenomenon of complementarity of DNA bases. With this method, small chromosomal defects can be detected or their paternal or maternal origin recognized.

Molecular studies that allow genotypes to be identified with greater precision, using Southern blotting methods (after digestion with restriction enzymes and polycridamine gel electrophoresis and subsequent hybridization with DNA complementary to the gene or DNA segments adjacent to it, with marked complementary DNA segments called " probes") or PCR (polymerase chain reaction that enables the amplification of segments of the problem DNA). Molecular methods can provide indirect information when the study is carried out on the DNA adjacent to and flanking the mutation being studied, the location of the gene and the distance between the mutation and the segments studied being previously known by linkage analysis. These methods are called indirect molecular, and for their study the molecular characterization of the affected individual and their relatives is essential, depending on the number of individuals to be studied and the relationship between them, and the type of inheritance with which the mutation is segregated.

The gene can also be studied directly, if its sequence is known and it has been previously cloned.
In this case, the molecular study is called direct and when there is little allelic heterogeneity this method can even be used to diagnose the disease, but not when there is great allelic heterogeneity, such as mutations for cystic fibrosis of the pancreas, in which case the clinical diagnosis is of particular importance.

Another method of molecular study is the sequencing of the mutation, of particular use in the previous case.

The application of molecular techniques has individuality in the approach to their use, depending on the characteristics of the defect, size of the gene and level of knowledge of the mutation and the basic defect, for each type of disease.

-Immunohistochemical methods are also used to determine the presence and characteristics of specific proteins, and in this way, establish a relationship between the mutation and its effect on gene expression. For this type of study, specific monoclonal antibodies are required.

IMMUNOLOGICAL FACTORS

In ancient Rome, all those people who were exempt from paying contributions were called immune. The observation that in some epidemics there were individuals who did not get sick introduced the term into medicine. Subsequent research showed that the immunological reaction was not only capable of defending against biological agents, but could also itself to be altered in various ways and give rise to illness.

When a healthy individual is exposed to different biological agents, he or she will be able to contract a disease, depending on the integrity of his or her immune system or apparatus, so immunity can be defined as that state that a healthy organism has that makes it not susceptible to contracting a disease.

However, in a broader sense, the immune system itself may be altered, due to a defect in its control and give rise to the disease; For example, autoimmune diseases.

Antigens and antibodies

An antigen is defined as that substance that is capable of awakening the reaction of the immune system, to cause the production of an antibody and for it to react with it. There are substances that do not have the capacity to induce an antibody by themselves, unless they bind to others.

These incomplete antigens are called haptens; they are usually lipids or simple carbohydrates, among others.

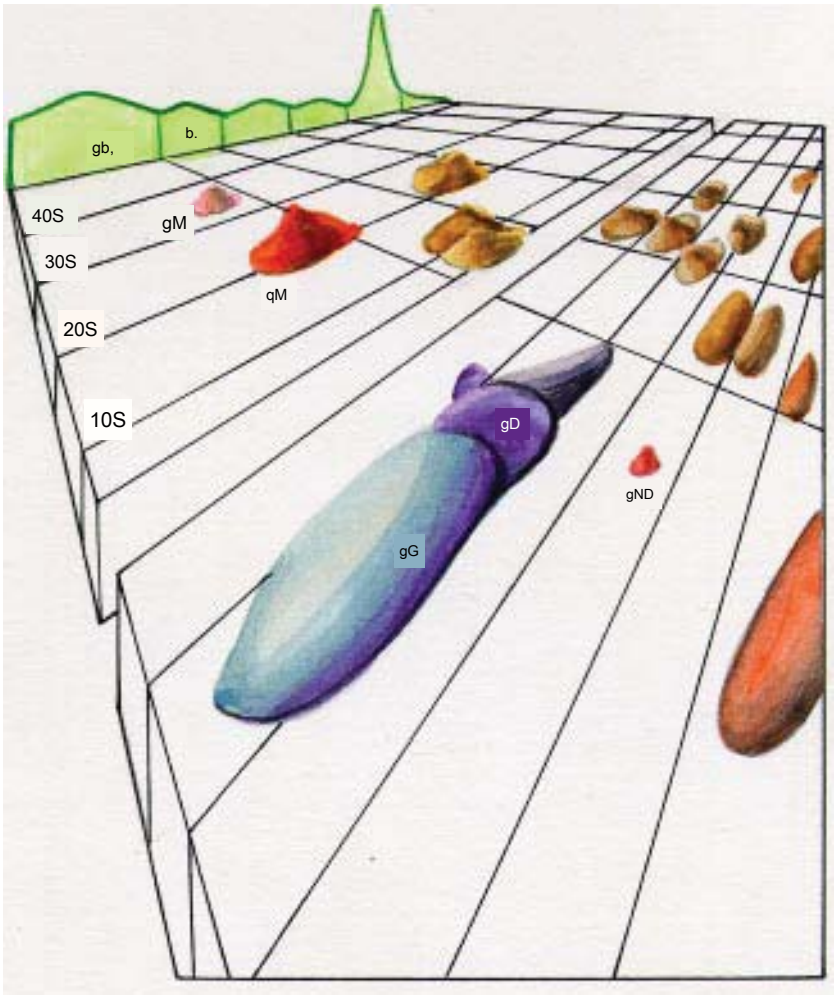


Fig. 22.7 Immunoglobulins.

Antibodies are proteins of the gamma-globulin type (immunoglobulins) that are produced as a response of plasma cells to antigen (Fig. 22.7).

structure of antibodies

Antibodies are made up of four polypeptide chains, two of them called heavy chains H (Heavy), which give them antibody specificity, and two light chains L (Light), linked by disulfide bonds.

The g(gamma) heavy chain corresponds to IgG:
IgM has the m (mu) chain; to IgA, the a (alpha) chain:
the d (delta) chain corresponds to IgD; and e(epsilon) to IgE.

The light chains are of the kappa type (k) or the lambda type (1).

In addition, there are subpopulations for immunoglobulins:

IgG, IgG, IgG, IgG,
IgA,, IgA,

There is an immunoglobulin found in the intestinal, bronchial, tear and other mucous membranes, with a different structure that makes it more resistant to the action of ferments called secretory IgA, which participates in local immunity.

-IgM has a molecular weight of 900,000, its ultracentrifugation constant is 19 S and it constitutes between 5 and 10% of the total antibody.

-IgG has a molecular weight of 160,000, its ultracentrifugation constant is 7 Sy and exists between 70 and 80%.

-Serum IgA has a molecular weight similar to IgG, representing between 10 and 20%, and shows an ultracentrifugation constant of 7-8 S.

Dynamics of the immune response

In figure 22.8 we can see two types of immunological reactions:

1. An innate non-specialized protection, not increased by repeated infections, which includes soluble and cellular elements, represented by: the integrity of the skin, the intestinal flora, lysozymes, complement (C1), phagocytosis by macrophages, NK cells and interferon, which together is called short-term efference.

2. Another protection mediated by a more specialized system, increased by repeated infections, called adaptive system.

In the graph you can see how the foreign agent called afferent is presented to other elements.

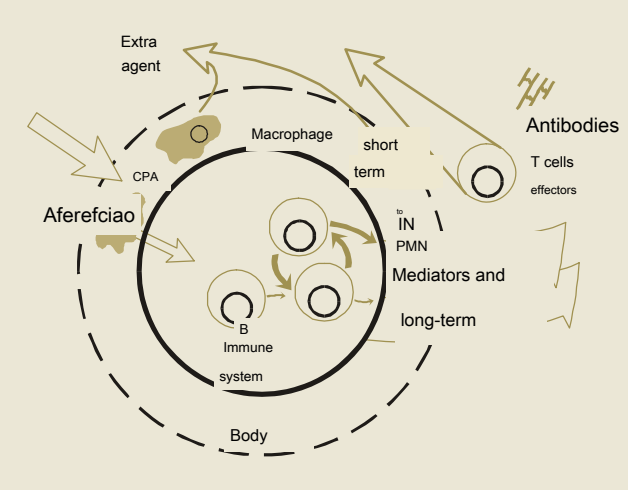


Fig. 22.8 Normal immune response.

ments (T and B lymphocytes), through the antigen-presenting cell (macrophage-APC).
This is defined as long-term efference, which includes not only effector lymphocytes, but also the product of their interaction with mediating antibodies, called cytokines or interleukins. This system is regulated by several mechanisms such as the major histocompatibility system (MHC), anti-idiotypes and others.

Immune reaction Its types

The immunological reaction encompasses all the phenomena that result from the interaction of specific cells of the immune system with the antigen, either through the production of antibodies (humoral immunity) or by direct cellular participation (cellular immunity) (according to the WHO classification, 1970, simplified).

That is, the immune system has two types of responses, one humoral, mediated by antibodies, and another cellular, mediated by cells, but in reality there is the interaction of both elements, which is called cellular cooperation.

For a clinical immunological reaction to occur, a prior exposure to the antigen (primary reaction) is necessary, followed by a variable period of time (latency period) and then a new exposure (secondary or anamnestic reaction).

Origin of the immune system

The current state of knowledge has allowed us to establish that (fig. 22.9):

1. Antibodies originate from plasma cells, dependent on B lymphocytes, and cellular immunity cells, fundamentally from T lymphocytes.
2. Ty B lymphocytes originate from the stem cell (in the bone marrow).

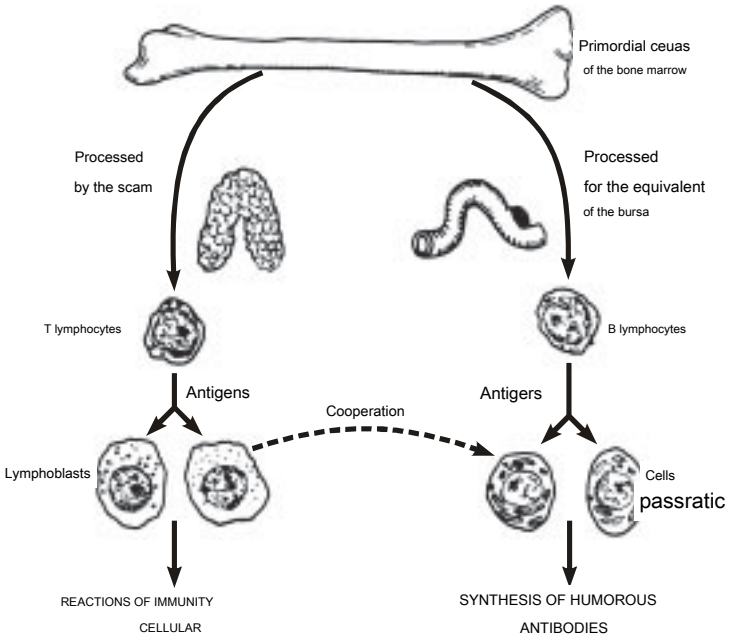


Fig. 22.9 Origin of the immune system.

3. The T lymphocyte is regulated and differentiates into different subpopulations, through the thymus, such as: T, (CD), T, (CD), T, (CD) and others.
4. The B lymphocyte is regulated by the bone marrow, in humans, and by the bursa of Fabricius in birds.
5. The term CD serves to express the differentiation cluster of each of the lymphocytes as they pass through the thymus.

Mechanisms of the immunological reaction

When the antigen penetrates, phagocytosis occurs, first by polymorphonuclear leukocytes and then by macrophages. This last type of phagocytosis is the one that interests us for the immunological reaction. The interaction of the antigen with macrophages modifies the cellular RNA or perhaps only combinations between fragments of antigens and RNA occur.

This RNA-antigen complex specifically selects the cell groups of lymphocytes genetically programmed to react with it. These lymphocytes then transform into immunoblasts.

If the antigen induces a humoral reaction, the activated lymphocytes will be type B and the immunoblasts will give rise to plasma cells that produce immunoglobulins (antibodies). On the other hand, if the reaction that occurs is cellular, the T lymphocytes transform into immunoblasts that will give rise to effector T lymphocytes.

humoral unit

Humoral immunity usually appears quickly and is carried out through the B lymphocyte system, for example. immunoglobulin-producing plasma cells. Immunity can be transferred from one subject to another using serum with antibodies (passive immunity).

IgM usually appears first in the primary reaction and IgG after IgM in the primary reaction and its presence signals the maximum intensity of the reaction; IgA appears later than IgG and its finding usually means prolonged rather than intense immunization.

In addition to its presence in plasma, it can be found in organic secretions, such as: tears, saliva, urine, and respiratory and intestinal secretions.

cellular unit

This type of immunity is carried out through T lymphocytes, it usually appears slowly, the cells act directly without the production of antibodies and the immunity can be transferred by sensitized cells (adoptive immunity).

Immunological reactions Classification

Gell and Coombs classified immunological reactions into five types, of which the first three and the last are mediated by immunoglobulins and the fourth, by cells.

Anaphylactic reaction

It is immediate. In it, the antibody (IgE) is coupled to the mast cell, and when it interacts with the antigen on the surface of the mast cell, it releases different substances that act as active chemical mediators (histamine, SRL, FQE and others). Examples: anaphylactic shock, bronchial asthma, urticaria and rhinitis.

Cytolytic reaction

Here the T lymphocyte (CD) participates, which reacts with different antigens to become effector cells and produces inflammation and the promotion of phagocytosis. Cytolytic reactions require the participation of complement. Examples: rheumatoid arthritis, Hashimoto's thyroiditis, transplant rejection (kidney and bone marrow), and contact dermatitis.

Arthus reaction and immune complex disease

In the Arthus reaction, the union of the antigen with the antibody produces microprecipitates around the small vessels, with the participation of complement.

In immune complex disease, circulating soluble complexes are produced secondary to the interaction of the excess antigen with the antibody in the circulating blood. These can be deposited in the vessels, joints and kidneys. This type of reaction also needs complement. Examples: serum sickness, glomerulonephritis, rheumatoid arthritis, and systemic lupus erythematosus.

Delayed hypersensitivity

The T lymphocyte (CD) participates in this reaction, which reacts with different types of antigens. It is described as cellular immunity. It is seen in the tuberculin reaction and in the rejection of allografts (homografts).

Type V reaction

This type of reaction occurs due to the introduction of antibodies that react with biologically active substances, canceling their function. Examples: antibodies against the intrinsic factor of vitamin B, (pernicious anemia); antibodies against the acetylcholine receptor at the neuromuscular junction (myasthenia gravis) and antibodies against the cell membrane (insulin resistance).

Importance of immunity in medicine

Immunity, as expressed above, can be a means of preserving health or a disease-producing mechanism.

Anti-infective immunity can be cited as an example of the first type and is defined as the specific defense capacity against a specific infection that is actively developed as a consequence of a previous contact with the causative agent, or received passively from another individual with active immunity.

As can be seen in Figure 22.10, the presence or absence of the disease will depend on the balance between the foreign agent and the response of the immune system.

The dynamics of the immune response is altered to cause disease, by the following mechanisms:

A. Due to response disorders (lack of response):

1. Hereditary. Agamma or hypogammaglobulinemia infantile
2. Acquired. Malignant hematological disorders (lymphomas): in the elderly; in AIDS.

B. Due to deposits of immune complexes, due to excess antigens: rheumatoid arthritis.

C. Due to autoimmunity. Production of antibodies against the organic components themselves not recognized as such by immunologically competent cells: collagen diseases; autoimmune hemolytic anemia; thyroiditis.

D. Due to hypersensitivity. Response to a normally non-antigenic substance: bronchial asthma; allergic dermatitis.

NEOPLASMS

A neoplasm is an abnormal mass of tissue whose growth exceeds and is incoordinated with that of the tissues

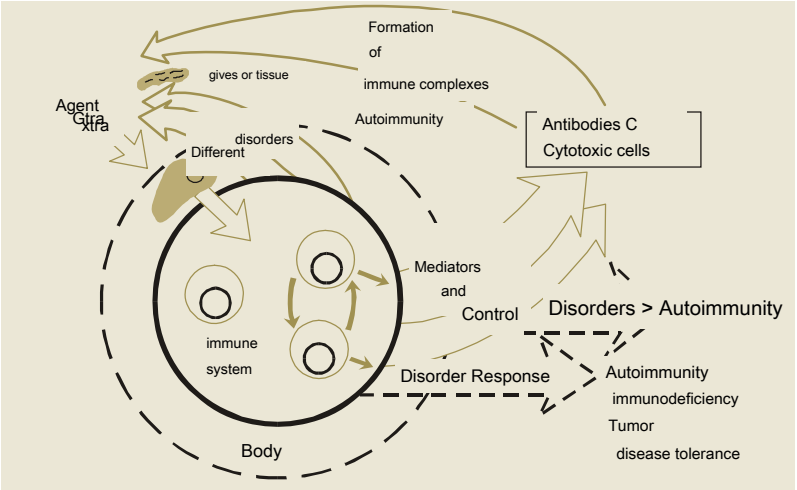


Fig. 22.10 Pathological immune response.

normal and that persists in the same excessive way, after the stimulus that triggered the change has ceased.

Differentiation and degrees

The degree of development and typicality of the cells is called differentiation. Greater differentiation in a neoplasm means that the cells are more similar to normal cells, and a tumor is said to be undifferentiated when its cells are more primitive and atypical. Four grades have been established, grade I corresponding to the most differentiated neoplasms and grade IV to the most undifferentiated.

Classification and nomenclature

Neoplasms are usually classified as benign or malignant, which generally presupposes prognostic inferences.

Benign neoplasms are made up of normal or almost normal cells, their growth is slow and local, their edges are clear, well delimited and they are frequently encapsulated. They do not show any tendency to spread to other organs.

Malignant neoplasms are formed by cells with varying degrees of atypicality, usually growing rapidly, without clear limitation of their borders, frequently infiltrating neighboring tissues and spreading to distant organs (metastasis).

Etiology

The etiology of the neoplasms is unknown. Current research seems to indicate a multiple causality; defined statistical relationships have been established with agents and factors of various kinds: chemical, physical, genetic, hormonal and viral.

Chemical agents

Percival Pott in 1775 described scrotal cancer in chimney sweeps; He related it to exposure to soot and suggested cleanliness as a preventive measure, which was proven in practice.

Currently, more than 500 substances are known to cause experimental cancer.

Multiple publications demonstrate the relationship between cigarette consumption and bronchogenic carcinoma.

Ryser in 1971 pointed out the following data when referring to chemical agents as producers of neoplasia:

1. The effects of carcinogens are dose dependent, additive and irreversible.
2. Carcinogenesis does not occur immediately, but after a variable period of time.
3. The changes caused by carcinogens are transmitted to the daughter cells.
4. Cancer can also be influenced by factors that are not carcinogenic.
5. Chemical carcinogens require cell proliferation.

Some proven carcinogens are listed below (Table 22.6).

Chart

age you	ecto organic
Tobacco	Oral cavity, pharynx, larynx, lung, esophagus and bladder
Alcohol	Oral cavity, larynx, esophagus and liver
Excessive diet Increased calories, fat and protein Asbestos	Breast, colon, gallbladder, endometrium
	Mesothelioma, lung
Benzene	Leukosis
Chromium	Lung
Isopropyl alcohol Vinyl chloride Alkylators	Perinasal sinuses Hepatic angiosarcoma
Azathioprine	Bladder, leukosis
Diethylstilbestrol	Lymphomas
Phenacetin	Vagina
	Kidney

physical factors

Exposure to sunlight in tropical and equatorial countries seems to favor the appearance of skin neoplasms, especially in white-skinned individuals. The frequency of this type of neoplasia among radiologists who regularly exposed their hands to X-rays was very high. Roentgen himself suffered from skin cancer. Irradiation of the thymus during childhood has been related to a higher frequency of thyroid neoplasms.

The high incidence of leukemia among survivors of the atomic bombings of Hiroshima and Nagasaki also suggests the importance of ionizing radiation in the production of neoplasms. Trauma, apparently, has no influence on the genesis of neoplasms.

It has not been possible to unquestionably specify the way physical agents act in oncogenesis, although most of the data seems to point towards a mutagenic effect on chromosomal DNA.

Hereditary factors

In humans, families have been observed in which a high frequency of neoplasms has appeared in up to four generations. There are some neoplastic diseases with a known family relationship: neurophy-

Bromatosis, colon polyposis, osteochondroma and retinoblastoma.

A three-fold higher relative risk has also been found in some families of suffering from breast, colon, stomach, endometrial, lung, malignant melanoma and ovarian cancer.

It remains to be determined how much corresponds to inherited genes and how much to environmental and behavioral influences.

Multiple intestinal polyposis, multiple endocrine neoplasias and Von Hipple-Lindau syndrome also recognize a hereditary origin.

There is an increased risk of leukemia in chromosomal disorders such as Down syndrome, Klinefelter syndrome, and Fanconi anemia.

Hormonal factors

Gonadotropins and estrogens induce neoplasms in the breasts, uterus and ovaries, but estrogens are useful in the treatment of prostate cancer.

The administration of androgens aggravates prostate carcinoma, but is used in the treatment of breast carcinoma.

In general terms, it is accepted that hormones do not initiate the oncogenesis process, but rather their function is catalytic.

Viral agents

Ellerman and Bang, in 1908, demonstrated the viral nature of chicken leukemia. Roux, in 1919, proved that sarcomas in some breeds of chicken could be caused by viruses.

Subsequent experiments confirmed that in both cases it was an RNA virus that was responsible for the disease. Oncogenic viruses are classified into RNA viruses (retroviruses) and DNA viruses.

In humans, the responsibility of retroviruses (RNA viruses) has been proven in some malignant tumors HTLV I and in HTLV II (Human T Leukemia virus) in adult T cell leukemias and some tricholeukemias, respectively.

The human immunodeficiency viruses HIV1 and HIV2, which cause the AIDS pandemic, belong to this class.

DNA viruses are also involved in the etiopathogenesis of different neoplasms (Table 22.7).

Chart

iru	eopla ia
Epstein-Barr (EBV)	Burkitt lymphoma Nasopharyngeal carcinoma
Epatitis B (BV)	epatoma
Papillomavirus	Cervix cancer, laryngeal papilloma

Pathogeny

At the time of secular transition, there is consensus in the scientific community that cancer is a disease of genetic origin: everything begins with damage, generally acquired, to a gene (mutation) due to environmental factors. This determines the clonal expansion of the single progenitor cell that suffered the genetic damage.

What does this damage consist of? It affects three normal regulatory genes: proto-oncogenes, anti-oncogenes and modulatory genes.

Proto-oncogenes are cellular genes that promote normal cell growth and differentiation. Due to the action of carcinogenic factors, oncogenes are created, the expression of which can lead to cancerous transformation of the cell.

An immune response includes so-called metastogenes and metasuppressor genes.

Once the mutations have been produced in the genome of the somatic cell (activation of oncogenes, inactivation of oncosuppressor genes or a combination of both), due to the effect of acquired environmental factors, conditioned, in turn, by behavioral and social factors, transformation, uncontrolled proliferation and clonal expansion of the affected cell occurs, causing the malignant neoplasm.

Oncogenes encode proteins, called oncoproteins, similar to the normal products of proto-oncogenes, except that they lack important regulatory elements and are independent of external stimuli.

The involvement of a single allele of the proto-oncogene is sufficient to initiate the neoplastic transformation, and it has a dominant character.

Antioncogenes are cancer suppressor genes;

Due to their normal expression, they inhibit the development of the cancerous phenotype and have a recessive character, since in order to allow the neoplastic transformation of the cell, both alleles must be inactivated.

Genes called modulators determine properties such as invasiveness, metastasization and the ability to generate neoplasms too frequently. They do not produce symptoms until advanced stages of their progress, which is tragic in many malignant tumors in which symptoms appear when the patient is invaded and curative treatments are not effective. This implies that the best measure is prevention, followed by rational and prudent screening for the disorder at the appropriate time, this should be as early as possible.

mechanical effects

They are the result of compression, invasion, or supplantation or ablation of normal parenchyma by tumor cells (mass effect) in situ or at a distance (metastasis).

It can cause pathological fractures in the bone; bone marrow invasion and myelophthisic anemia.

Compression of normal structures and destruction of tubular organs are observed in abdominal neoplasms, occlusive syndromes, obstructive jaundice, and ureteral or bladder compression. Atelectasis of bronchogenic carcinoma is classic. Expansive brain lesions are expressed by focal symptoms and intracranial hypertension.

Non-metastatic distant effects

Some malignant or benign tumors located in endocrine organs can show functional activity, such as pituitary, thyroid and adrenal adenomas, to name the best known.

Certain malignant tumors produce ectopic hormones such as lung cancer: ACTH, PTH, ADH, among others. Others exhibit non-metastatic effects not attributable to hormones, at the level of different devices and systems (Table 22.8).

Cachexia-anorexia syndrome usually occurs at some point during the course of uncontrolled cancer. It is postulated that it is due to multiple factors such as abnormalities in taste and smell, with decreased caloric intake, protein losses associated with spills and hemorrhages, digestive dysfunction (obstruction and malabsorption), hypermetabolism and effects attributable to treatment (nausea and vomiting). Factors secreted by the tumor have been identified, such as a TNFL called cachexin that inhibits certain lipogenic enzymes with consumption of fat reserves.

There are authors who add psychosocial effects, considering the profound repercussions of the disorder on the patient's psyche and their family, work and community environment.

Diagnosis of neoplasms

A complete clinical history with detailed anamnestic data and a careful physical examination are essential elements in the diagnosis; However, this can be more useful in relation to benign tumors and precancerous diseases (oral leukoplakia, for example) than with cancer itself, since in many cases by the time the clinical stage is reached it is already very advanced.

It is good to remember at this moment what has been called the "constellation of the four A's", given by the association of anorexia, asthenia, weight loss and anemia, since most of the time this group means malignant neoplasia.

Laboratory, endoscopic and radiological studies are indicated depending on the location of the neoplasia.

Chart

i theme	drome	eca i mo pri cipal	Ocali aci
Endocrine	Cushing	ACT	Lung
	YES. A.	A.D.	Lung
	Ipercalcemia	P.T.	Lung
	Gynecomastia	Chorionic Gonadotropin	Lung
Highly strung	Myasthenic	Autoantibodies Lung	Lung
	Cerebellar degeneration	Breast Various	Autoantibodies
	Polyneuropathy		
Skin and SOMA	Acanthosis nigricans		Several
	Dermatomyositis	Autoantibodies Various	
	erythroderma		Lymphomas
	Osteoarthropathy pulmonary hypertrophic		Lung
emolinfopoietic	Thrombophlebitis migratrix	Substances thromboplastic Various	
	Endocarditis	Thromboplastic	
	marasmatic	substances Various	
	Polyglobulia	Erythropoietin Kidney	
	Anemia	Various	Several
Renal	Nephrotic syndrome	Immunological	odgkin

The certain diagnosis of a neoplasia is supported by the histological study of it by means of a biopsy or by the cytological analysis of secretions (exfoliative cytology).

PSYCHOLOGICAL FACTORS

Psychological factors play an important role in the appearance and development of different disorders common in modern life. They will be studied in the corresponding subject.

IATROGENIC FACTORS

The definition of iatrogenesis was already stated in Section I, "Introduction to the Clinic".

Nowadays, medical care has achieved great preponderance and influence on people and society as a whole. Scientific-technical development in the field of medicine has contributed to raising the level of health of the population, particularly in societies where medical care is guaranteed to the entire population. There is also full awareness that medical care can also cause illness.

Without attempting to exhaust the study of this formidable phenomenon, we will briefly point out the main mechanisms involved in the iatrogenic process:

I. Due to iatrogenic doctor-patient relationship: defect in communication, lack of tact or prudence on the part of the doctor; alarmist rather than reassuring attitude.

II. Lack of preventive sense on the part of the doctor, who may miss opportunities to educate or protect his patients and thus avoid the subsequent development of preventable diseases. Examples: ischemic heart disease, tetanus and parasitism.

III. By diagnostic procedures, in particular, those that involve invasive techniques. Examples: vascular and cavity punctures, biopsies and angiograms.

IV. By therapeutic procedures.

A. Blood transfusion:

1. Transfusion reaction.
2. Disseminated intravascular coagulation.
3. Hepatitis due to By C virus.
4. AIDS.

B. Injections:

1. Tetanus.
2. Hepatitis due to By C virus.
3. AIDS.
4. Intramuscular abscess.

C. Ionizing radiation (see physical factors).

D. Surgical interventions:

1. Immediate complications.
2. Late complications.

E. Drug toxicity (see table 22.2):

1. Digitalis poisoning.
2. Allergic reactions.
3. Spinal cord inhibition by drugs.

F. Side effects (see table 22.2):

1. Therapeutic Cushing.
2. Superinfections due to antibiotics.
3. Bleeding due to anticoagulants.

In short, it is not about renouncing advances in science and technology, but about using them with prudence and moderation. We must reinforce the preventive sense of medical care, the educational action of the health team. Do not lavish investigations, medications or operations, but only indicate them when they are scientifically founded and there is clear evidence that the patient can obtain a net benefit from this procedure.

MEDICAL INTERVIEW.
CLINICAL HISTORY. ANAMNESIS

23

MEDICAL INTERVIEW

The medical interview, or better the doctor-patient interview, constitutes a unique model of human communication and serves as a framework for the intellectual and emotional interaction commonly known as the doctor-patient relationship.

It is up to another discipline (Medical Psychology) to deal with the study of the doctor-patient relationship. Here we will only refer to the interview technique for preparing the clinical history.

The doctor-patient interview considers formal and content aspects, to which it is necessary to pay attention. At all times, the doctor must be welcoming, affectionate and respectful towards the patient. The human relationship should never be subordinated to the purely technical one.

From a formal point of view, the doctor must aim to achieve optimal communication with the patient, which favors not only the preparation of the medical history, but also the fundamental objectives of medical care, which are: preserving the health of the healthy man and recovering and rehabilitating the health of the sick man.

The first interview is of particular importance. The patient does not know the doctor and has certain expectations, depending on his personality, cultural level, and psycho-emotional state. When you enter the office, the doctor must look you in the face, greet you and smile at you. Since then, he must always call him by his name. Invite him to sit.

There are those who recommend not placing the consultation table, as a barrier, between them, but rather sitting the patient on one side of it.

The interview should begin with an open question, which gives rise to an uninterrupted story about the health problems that led you to attend the consultation. The question may be of the following type:

How do you feel? Or, How can I help you? Or What problem do you have? It is essential to ask the question in a thoughtful, kind and persuasive tone. Next, the doctor must know how to remain silent, without interrupting the patient in his story; Pay close attention to it, without showing impatience or boredom. Once you finish, you can stimulate it or continue with another generic question such as: what else?, which can be repeated, if it is estimated that there is still important data not referred to. He then proceeds to ask questions directed according to the problems mentioned and other rigorous questions to specify the patient's personal and family history. The doctor must show this

special interest in preventive aspects such as immunizations, harmful lifestyle habits and hygienic habits, since this attitude, in itself, constitutes an important educational factor.

The questions are formulated in an objective, impartial, non-biased manner, so that they do not induce or contaminate the answers. Language that is clear and understandable to the patient should be used. A climate of security and discretion should be fostered that encourages frank analysis of any health problem, including those of a more intimate nature.

Carrying out the physical examination is inherent to the interview. A good doctor-patient relationship is fostered by performing a good physical examination and this, in turn, reaffirms the patient's confidence in the doctor's ability.

Complementary exams are indicated below, if necessary. When the required data is available, the doctor reaches diagnostic conclusions and draws up a therapeutic plan. It also formulates its prognostic assessments including possible complications.

From the first consultation, it is necessary that communication also be established or flow from the doctor to the patient, so that the health problems that have been detected, their nature, the need or not for complementary examinations and the foundations of the prevention, cure and rehabilitation measures that should be undertaken are explained.

Human communication is not only established through words (Pavlov's second signal system). There is also numerical communication or extraverbal language (first signal system). No less relevant is the double content that every message contains: the manifest content, represented by what is literally or formally said, and the latent content, with a greater subjective connotation, which does not always coincide with the previous one and often has different or opposite meaning.

When communicating with the patient, the doctor must pay close attention to his extraverbal language and be very careful to match both contents of the message. At all times, the doctor must reassure the patient, instill confidence in his recovery and convince him to adopt the treatment he proposes.

The technical content of the interview as far as the preparation of the clinical history is concerned, is represented by the anamnesis and the physical examination.

CLINICAL HISTORY

Before beginning the medical history, we must point out some aspects related to medical care.

Medical care can be understood as the set of actions or care provided by the doctor and other hierarchical technicians that tend to promote, recover or rehabilitate the health of the individual, the family and the community.

Our national health system guarantees qualified medical care to the entire population. According to their nature, health problems can be addressed by three levels of medical care:

Primary care. It constitutes the patient's first contact with the health system, that is, their gateway. It is provided in the community where the patient resides, by the health team of the polyclinic0* or dispensary. It has an outpatient, consultative or domiciliary nature. It includes the healthy and the sick; to those who request attention and to those who do not.

The community medicine model ensures comprehensive medical care delivered** and continued by the same health team (doctor and nurse) to each sector of the population.

Secondary care. It is the one provided in a second step, to which the user does not have direct access, but rather through a referral from the primary care doctor. It can be outpatient (in the polyclinic itself or in external hospital services) or inpatient.

Tertiary care. It is one that, due to its very specialized condition, is only provided in provincial or national centers, such as, for example, neurosurgery, cardiovascular surgery, kidney transplantation, burns and other services, including research institutes.

We can now deal with the clinical history by first pointing out what it consists of and then immediately analyzing the clinical history in primary care (that is, in the community polyclinic) and the clinical history in secondary care (that is, in the hospital), since although the same in their essence, they present some characteristics that are worth pointing out.

The criteria that will be stated here correspond to our national health system.

The clinical history serves to make an orderly collection of symptoms, signs, identity data and others, which allow the doctor to propose a clinical, syndromic and nosological diagnosis. This diagnosis may be in its first provisional phase, and is affirmed or denied with the analysis of the results of laboratory investigations.

* Polyclinic: Term used to designate the polyclinic in Cuba.

¨ Dispensarized: Special attention on a scheduled basis, which is provided in polyclinics to certain diseases.

clinical, radiological, endoscopic or other types of complementary calls.

The fundamental objective of the Clinical Propedeutics subject is to teach the medical student to prepare a good clinical history. This should be called a better clinical record, although use has consecrated the first name.

CLINICAL HISTORY IN PRIMARY CARE

The clinical history is the basic document of primary medical care. It can be considered as the methodological guide to comprehensively identify the health problems of each person and establish not only the needs they feel, which lead them to request medical consultation, but all their needs. In addition to this diagnostic function, the clinical history serves as a basis for the planning, execution and control in each case of actions aimed at promoting, recovering and rehabilitating health.

In this way, the clinical history in primary care becomes an effective instrument to apply comprehensive medical care programs to people in their family and social context, which aims to achieve personal and environmental health, protect the population, particularly vulnerable or at-risk groups, prevent and control the main causes of mortality and morbidity in different age groups, through the identification and control of contributing or predisposing factors in healthy (promotion) and sick people. (rehabilitation).

The clinical history in primary care consists of the anamnesis and physical examination. It also includes diagnostic and therapeutic aspects (conduct to follow).

Anamnesis

The anamnesis includes the following sections:

1. Personal identity data.
2. Reason for consultation.
3. History of current illness.
4. Life habits.
5. Family history of illness.
6. Gynecobstetric history.
7. Personal history of illness.
8. Operations.
9. Immunizations.

Personal identity data includes age, sex, origin, occupation and education.

Age and sex are important in relation to the preferential appearance of many diseases.

The origin can be valuable information for the diagnosis of disorders related to hygienic-epidemiological environmental conditions.

Occupation is important in the investigation of occupational risk or illness. In this sense, it is necessary to record the type of work that the patient does and the working conditions of their center.

Education as an indicator contributes to shaping the sociocultural profile of the patient, frequently linked to certain diseases facilitated by ignorance and lack of culture.

The reason for consultation must express the main motivation that leads the patient to request medical attention and when this is not the case, in cases where the initiative comes from the polyclinic, the medical reasons that determine it. For example, the patient may come because of fever, or diarrhea, or chest pain.

The doctor can summon the patient for a consultation or visit him at home to carry out a scheduled medical check-up on diseases subject to general care such as high blood pressure, diabetes mellitus, severe bronchial asthma, pulmonary tuberculosis, among others. They can also be healthy people, in whom a health check is carried out for fundamentally preventive and educational purposes, such as: pregnant women, newborns, infants, preschoolers and schoolchildren, adolescents, the elderly, workers at occupational risk and others.

The history of the current illness should briefly and concisely include the fundamental features of the health problem presented by the patient. Strictly speaking, it is not always a disease. As we have pointed out, a healthy person can be treated to control disease risks, through health promotion and specific protection actions.

The registry of lifestyle habits allows us to identify the main risk factors for chronic non-communicable diseases: smoking, sedentary lifestyle, dietary habits and emotional tension. Its simple investigation is already instructive for the patient and serves as a basis for undertaking further educational actions aimed at modifying these harmful habits, both in healthy people (health promotion) and in affected subjects (rehabilitation).

Family history of disease is important in the investigation of hereditary diseases (such as bronchial asthma, diabetes mellitus, sickle cell anemia), communicable diseases (such as pulmonary TB, leprosy, infectious hepatitis) and in health-cultural-based disorders (such as parasitism, nutritional disorders due to deficiency and excess).

Perinatal history (prenatal, natal, postnatal) can be valuable especially in children.

The vaccinations received must be specified with a view to updating them, taking into account the current immunization program.

The gynecobstetric history should include menstrual history: menarche, menstrual formula, date of last menstruation. Obstetric history:
pregnancies, births, abortions (spontaneous or induced), date of last birth. With the necessary prudence and tact, aspects of medical interest regarding sexual relations can be addressed: age of first relations, frequency, promiscuity, contraceptive methods used and, when appropriate, quality of sexuality (orgasm, technique used, expectations), culture and mental patterns in this regard.

Pregnancy and its follow-up entail special control and clinical history, as well as the assessment of perinatal risk in women.

The performance and results of the cervical cytological test must be recorded.

The personal pathological history allows us to identify both the diseases suffered and those that the patient may present. It is common for a sick person not to know that he or she is sick (hidden morbidity), because the disorder does not cause sufficient subjective discomfort. Population morbidity studies have shown that the hidden part of the iceberg (hidden morbidity) is much greater than the exposed part (declared morbidity) in numerous diseases, such as arterial hypertension, ischemic heart disease, diabetes mellitus, urinary infection, anemia and others. investigation, the disease could remain undetected, depriving the patient of the benefits derived from early and timely care.

The operations performed on the patient give insight into the diseases that caused them, as well as any possible post-surgical sequelae.

Physical examination

The physical examination in primary care conditions must necessarily be brief, without overlooking a regional and overall assessment of the patient.

Schematically, the physical examination can be framed in three moments or stages:

First stage. With the patient sitting on the stretcher. The head is explored, including facies, ocular conjunctivae and oral cavity. Neck, checking for thyroid enlargement or other tumors, as well as jugular engorgement. Thorax and upper limbs, with measurement of temperature, respiratory rate, radial pulse and blood pressure.

Second stage. With the patient in a supine position on the stretcher. Auscultation of the heart is performed. It is explo-

Abdomen, including screening for hernias and hepatosplenic enlargement or other tumors, and lower limbs, including palpation of pedal pulses and screening for foot deformities. Examination of the breasts and respiratory system. Psychomotor development is examined in children; In women, the gynecological examination should always be performed in the presence of a nurse.

Third stage. Intended for weighing and measuring the patient with a view to evaluating his/her stature (obesity and malnutrition screening). Preferably the weight should be expressed in kilograms and the height in centimeters.

Apart from the anamnesis and physical examination, the clinical history in primary care also includes the main complementary investigations that, depending on the case, should be carried out. Its design allows establishing the diagnostic problems detected, including risk factors, as well as specifying the conduct to be followed regarding measures aimed at health promotion, recovery and rehabilitation. Finally, the clinical history in primary care serves as a framework to evaluate the patient's evolution, the control of dispensation and the results of the health programs applied.

CLINICAL HISTORY IN SECONDARY CARE

In secondary care in the hospital, the clinical history includes other elements, since the patient can be re-examined and evaluated daily and even several times a day if required, in addition to having more complex exploratory methods that allow the diagnosis to be better specified (see models at the end of the chapter).

With the daily examination of the admitted patient, the evolution of the disease is monitored. A first detailed evolution in which the diagnostic impression is captured, and a diagnostic discussion elaborated with all the elements that help to propose the different types of diagnoses, are essential in this part of the clinical history.

All of the above makes it possible to institute the appropriate treatment, which will be reflected in the instructions written by the doctor. Interconsultations with different specialists will also be written down if needed.

From the moment of admission to the hospital, a series of data are collected in the emergency department and shown in the general medical history. This is where the first instructions are given before being sent to the hospitalization room.

On a discharge sheet that is placed at the end of the clinical history, the final diagnosis is written, the procedures

diagnostic and therapeutic procedures used. In case of death, the necropsy protocol will be added.
A copy of this discharge sheet must be sent to the polyclinic to which the patient belongs for follow-up, which will provide an adequate polyclinic-hospital relationship. All the aspects indicated in this graduation sheet will be ordered in such a way as to enable statistical studies by computer.

Once archived, this clinical history allows its use in cases of patient readmission or to carry out scientific research on morbidity, mortality or any other type of medical work.

It follows the enormous importance of preparing this file, which must reflect the biopsychosocial reality of the patient.

The two fundamental factors of the clinical history in secondary care are also: the interrogation and the physical examination.

Interrogation

Through the interrogation, the following data is collected:

1. Personal identity.
2. Reason for admission or consultation.
3. History of current illness.
4. Personal pathological history.
5. Family pathological history.
6. Toxic habits, environmental data of interest and others.
7. Psychosocial history.
8. Interrogations by general systems and symptoms.

Physical examination

1. General physical examination.
2. Regional physical examination.
3. Physical examination by systems:
   a) Osteomyoarticular system.
   b) Respiratory system.
   c) Circulatory system.
   d) Digestive system.
   e) Genitourinary system.
   f) Hemolymphopoietic system.
   g) Endocrine system.
   h) Nervous system:
      Neurological examination.
      Psychic exam.

In Section I of this work, the basic examination techniques were already discussed, in Chapter 3, and the examination techniques of the physical examination in a supposedly healthy individual, in the rest of the Section; In this chapter we will insist on the interrogation, and in the rest of Section II the clinical examination of the sick individual will be detailed.

Through the interrogation we obtain the data that corresponds to personal identity and the current illness; This is what is called proximate anamnesis, as well as personal history and family history or remote anamnesis.

Before delving into the data collected by the anamnesis, we do not want to overlook the importance of specifying the psychosocial history, since man is a biopsychosocial being, and many conditions would fail to be diagnosed if these factors that so influence the reasoning process are not well known. If these factors are ignored, we would not be able to establish a real diagnosis.

It must be insisted that it is essential to collect both the somatic, psychological and social aspects, therefore, a good clinical history is one in which a good biography of the patient is obtained and which must take into account:

- Birth and psychomotor development.
- Data on preschool and school ages.
- Educational history.
- Occupational history.
- Psychosexual and marital history.
- Social adaptability.
- General activities and interests.
- Previous psychopathological medical history.
- Socioeconomic history.

ANAMNESIS OR INTERROGATION OF THE DISEASE

The anamnesis (from the Greek anamnesis: appeal to memory, remembrance, from ana: again and mnesis: memory), also called commemorative or antecedents, includes all the data that we obtain by questioning the patient regarding the symptoms of the current illness, the patient's previous illnesses, his diet, lifestyle, etc., and the illnesses in his family.

We must distinguish between proximal anamnesis and remote anamnesis. The first includes the data on the illness suffered by the patient at the time of the examination, what we call the current illness; The second refers to all the previous data, that is, to the pathological past of the patient (personal pathological history) and the different diseases observed in his family (family or hereditary pathological history).

The interrogation is the exploratory resource that we use to obtain anamnestic data and with it we begin the clinical examination of the patients.

It consists, as its name indicates, of asking the patient - or his or her closest relatives - about the different symptoms of their illness, as well as the other data that make up the anamnesis.

Even though the value of the interrogation is not always as great as that of other exploration resources, it should be known that it provides valuable diagnostic elements and that, sometimes, it is the only one that provides the elements to base the diagnosis. Therefore it will be used whenever possible.

As is understandable, interrogation can only be used in patients of a certain age, of normal intelligence, of good will and with means of expression. Limited or disturbed intelligence, and certain ages, make the practice of interrogation difficult or impossible, as does the lack of means of expression (foreign languages). The good will of the patient and his cooperation are essential, and the possibility of simulation or concealment of illnesses must always be kept in mind.

As general rules for carrying out the interrogation, the following can be mentioned: order and clarity in the questions, respect for social and personal conveniences, education and delicacy, so as not to hurt modesty, or create embarrassing situations, sometimes very serious;
discretion and reserve. The questions must be pertinent, intentional, avoiding asking superfluous and, above all, unnecessary questions, such as asking someone who is dyspneic at rest if they have difficulty breathing when walking, for example. It is unnecessary to remember that in serious cases, where urgent therapeutic action must be taken, the interrogation must be dispensed with, for the moment, or reduced to quick and concrete questions of obvious usefulness.

Interrogating well is very difficult and requires complete knowledge of senmiology and medical and surgical pathology, backed by good clinical experience and a great knowledge of men.

The seasoned clinician listens patiently to everything that is said to him and believes what the circumstances warrant and the physical examination corroborates. It has been well said that "an interrogation requires the strategy of a diplomat and the tact of a confessor."

NEXT ANAMNESIS

The interrogation begins by taking personal identity data, to continue with those from the immediate anamnesis, that is, what seems most important to the patient, and ends with the study of the other diagnostic elements of the remote anamnesis.

Many times, the anamnesis is so characteristic that it almost alone allows the diagnosis of the disease.

Other times it is poor in data, without being able to extract she any guidance. Hence, from the point of view of their diagnostic value, we can distinguish typical anamnesis and confusing anamnesis. The first, as their name indicates, are of the greatest diagnostic importance; the latter, of very little or no interest.

When a patient tells us that, being in good health, he was suddenly overcome by a strong chill, followed by high fever, with intense pain in the chest (side pain), respiratory difficulty (dyspnea) and special reddish expectoration, he provides us with such a typical anamnesis that the diagnosis of pneumonia can almost be established without further examination. Likewise, the diagnosis of renal lithiasis, with renal colic, would arise from a history in which very intense pain was reported in the lumbar region, radiating along the path of the ureter, with painful spread to the testicle on the same side and the glans penis in men, or to the corresponding labia majora in women, followed by the emission of bloody urine. Thus the examples of typical anamnesis could be multiplied.

On the other hand, as sometimes happens, either due to individual conditions of the patient, or due to the nature of the disease itself, confusing anamnesis may be found, which does not help us at all in the diagnosis.
This is the case when symptoms that we have called common or banal are collected, such as general malaise, lack of appetite, weight loss, headache, etc., which can correspond to very varied morbid processes.

The interrogation requires, as we have said, complete knowledge of the symptoms of all diseases. Therefore it can only be done with some benefit, at the end of our studies.

We must now consider the importance of the clinical history data collected by the interrogation.

In addition to finding here the symptoms for which the patient is consulted, the date of their appearance, the evolution followed, their relationships with other symptoms - all elements of the highest interest for the diagnosis -, we can discover certain etiological factors that have to contribute, many times, not only to clarify the nature of the patient's illness, but also to start us on the path of causal or etiological diagnosis.

With some frequency, the patient tells us what he assumes is responsible for his illness, that is, the alleged cause of the illness. Even though, on numerous occasions, this supposed cause is not related to the true etiology of the pathological process, in others it constitutes important information, such as when it comes to contacts of the patient with individuals carrying an infectious disease, for example, or when it clarifies the cause of a digestive disorder.

requiring the ingestion of food in poor condition or that is very difficult to digest. Chilling is a presumed cause of illness that enjoys great favor among laymen, thus it will be seen in practice, with some frequency, that patients accuse temperature changes, especially air currents, as responsible for their illness. In fact, cold can play an important etiological role in some cases. Thus, among cold diseases, that is, due to cooling, we have paroxysmal hemoglobinuria, cold facial paralysis and catarrhal processes of the upper respiratory tract. Cooling acts, rather, as a contributing or occasional cause.

Likewise, other etiological factors can be found here, such as trauma, poisoning, diet, habits, etc., to which we will refer immediately, when considering the diagnostic interest of the different sections of the clinical history.

Personal identity data

We first consider data that have only administrative and identification interest: name, address and nationality. Not so the naturalness, which may present diagnostic interest, and will be studied, so as not to incur repetitions, with the origin.

Profession

It acquires relevant importance as an etiological factor in many occupational diseases. Naturally, in these cases its diagnostic value is great, often decisive. For this reason, it is noted in the clinical history and carefully investigated, not only specifying the patient's current occupation, but also previous ones, if they exist.

Among the most important occupational diseases we can point out lead poisoning or lead poisoning, suffered by those who manipulate, in one way or another, this metal, responsible for many high blood pressures, paralysis due to polyneuritis, intestinal colic - called lead colic -, etc.; pneumoconiosis, in stonecutters: calicosis or silicosis, in coal miners: anthracosis, with its subacute or chronic bronchopulmonary symptoms;
varicose veins of the lower extremities in those who work standing (stomatologists, shop assistants) and obesity in sedentary professions. The frequency with which people subjected to great emotional stress, including doctors, suffer from myocardial infarction should be mentioned.

dad

Of great importance as an etiological factor (predisposing cause), it sometimes reaches diagnostic value.
We can point out that gastrointestinal disorders are common in childhood, often due to bad eating habits.

It should be noted that infant morbidity and mortality rates have decreased considerably in our country. At the beginning of the 1960s, health programs and activities began to be carried out in Cuba, which quickly had a favorable effect in reducing infant mortality. This was also achieved by the important economic and social development that began in the same period as a consequence of the socialist revolutionary process that began on this same date.

Already in the early years of the 1960s, malaria, poliomyelitis and diphtheria were eradicated, other diseases have been reduced to a minimum.

In Cuba, nutritional diseases have almost disappeared; However, these present a high prevalence in underdeveloped countries, where the operating factors are very varied and are particularly established in the sectors of the economy, agriculture, health and education. Due to them, nutritional problems are closely linked to the development of the country and cannot be framed in isolation in health programs, but rather as part of a multisectoral program, which addresses the different causal factors at the same time. Rickets, other vitamin deficiencies and deficiency diseases are still seen wreaking havoc in many countries.

Many eruptive diseases occur more frequently in second childhood, which is explained by the receptivity of this stage of life and the opportunity for contagion.

During puberty, disorders related to the development of sexual functions are found.

In mature age the struggle for life and the full enjoyment of vital functions weighs heavily. Nervous disorders, venereal diseases and metabolism are observed here. Cancer is more common after age 40, although it can appear from an early age in life.
In old age we find, above all, atherosclerosis and its complications, pulmonary emphysema and prostatic hypertrophy, among

Sex

Apart from the diseases that affect the genital system, naturally different in each sex, this information is of interest due to the semiological value it acquires in some cases. In fact, sex seems to influence, in a certain way, the appearance of certain diseases that statistics show with a clear predominance in one sex or another.

Thus we can point out the greater frequency of exophthalmic goiter, hysteria, chorea, hemicrania, obesity and gallstones in women. On the other hand, certain diseases of the skin are much more common in men.

nutrition (diabetes and gout), pernicious anemia, aortic lesions, etc.

Race

The influence of ethnic factors on the etiology of diseases justifies taking them into account in the clinical history, where they sometimes acquire a certain semiological value.

Sicklemia (anemia caused by a hemoglobin disorder) occurs in the black race. In the white race, the predisposition of the Anglo-Saxons to gout and scarlet fever should be noted, as well as thromboangiitis obliterans and metabolic diseases among the Hebrews, ischemic heart disease among the Finns, and hepatitis among the Czechs.

civil status

It also offers diagnostic interest, especially in women, whose genital conditions depend in a high percentage on sexual relations, normal or ectopic pregnancies, childbirth, abortions, etc.

Origin

It is of great interest and in many cases helps in the diagnosis, especially in determining the origin of the disease. Its importance rests mainly on the occasion of contagion. Thus, when studying a liver tumor, the fact that the patient is natural or comes from a region or country where the hydatid cyst exists, will cause this disease to be seriously considered among the different diagnostic possibilities. The same can be said of patients coming from malarial, goiter areas or those where intestinal parasitism, typhoid fever and other infectious diseases exist.

Reason for admission

It refers to the brief notation of the symptoms that make the patient consult, and the patient's own words must be written. It must consist of one or several words or one or two phrases. For example: "headache", "shortness of breath", " diarrhea", "chest pain", "vomiting blood", etc. Another doctor's diagnosis or interpretation of symptoms should not be included in the reason for admission, and terms such as " duodenal ulcer", "gallbladder lithiasis", "high blood pressure", "diabetes", "hematemesis", "melena", etc. should be avoided.

history of the current illness

This chapter of the medical history represents a compilation of the complaints that the patient brings to the doctor and expands the data on the reason for admission. This part is the one that requires the most art in the making of the story. You need knowledge of the disease, which can only be obtained with experience. Thus, a judicious interrogation will give us the details of the patient's complaints.

Only a few patients are such good observers that they will be able to give us the complete history of their current illness without the need for directed questioning. When we encounter a patient who gives us a good, detailed history, we usually suspect that the patient has read about their symptoms or has already consulted other doctors, who have asked them questions.

As an example let's consider a patient with a peptic ulcer. It is likely that he will tell us that he has a "pain in the pit of the stomach" (abdomen) and that he has "gas" and that he belches. The doctor, knowing the symptoms, will be able to obtain by asking questions that epigastric pain occurs in episodes over a few days, weeks or months; that it appears at a certain time after meals; that it is relieved by the ingestion of food and alkaline; that it can wake the patient up during night: and that can be localized or can radiate directly to the back.

The beginner can only learn by doing stories.
Thus, you must follow a plan, more or less as follows:

The patient should be asked about the approximate date of the beginning of his or her reason for admission or consultation, either specifically or in terms of three weeks ago, "six months ago or "one year ago. It is also valuable to note what the patient considers to be the cause that led him to the consultation or that may be the reason for admission. After this, we must ask you to describe the evolution of your symptoms or complaints. The questions that should always be used until the syndromes are known are: What? Where?

When? How was it modified, changed, or alleviated? Because? For example: the "that" can be a pain; the where", the site of the pain and its irradiation; the when", the moment of the pain; "how it was modified, changed or relieved and what relieved it", the relief of pain by ingesting food or bicarbonate in the case of abdominal pain; aggravation of chest pain from breathing; increased back pain when bending, and other questions along these lines.

If the disease is characterized by similar recurrent attacks, only one of them needs to be described, and any variations or complications that may have occurred should be noted. It is often best to obtain a description of the last episode, as it will be the one that is freshest in the patient's memory.

One of the most difficult things to convince students is that certain negative data can be, and usually are, of as much or even greater interest than positive data. For example, if the reason for admission or consultation is chest pain, it may be

It is of great importance to know that pain is not increased by inspiration. It may be important to know in the case of a headache that the use of vision for close work is not the cause of this symptom.

Many patients tend to deviate from the history of their current illness, and the historian must, through questioning, guide them through the logical development of their history. This can be done more easily by taking the patient through a chronological account of their symptoms.
This simplifies history taking and creates a better clinical picture if a chronological plan is followed. This attitude, indispensable in the history of current illness, is what leads us to what we know today as a chronopathogram.

The chronopathogram will, therefore, be the evolution, over time, of the different symptoms that constitute the patient' s illness, noted in a careful and orderly manner.

Questioning by systems

After obtaining as much information as possible by the aforementioned methods, the student should review the different systems in search of evidence of disorders in each sphere, related to the current illness. Patients frequently do not associate symptoms that seem irrelevant with their reason for consultation or admission. Only through interrogation can we obtain these symptoms and their associations. We can illustrate this very well in the case of thyrotoxicosis, for example, where several systems may be affected. The reason for consultation in this case may be 'nervousness', 'fatigue' and 'weight loss'.

As part of the current illness, the interrogation may reveal the following:

1. That vision is abnormal and that the eyes have become more prominent.
2. That swallowing is sometimes affected and that the throat feels full.
3. That the voice is deep and that there is difficulty breathing when exercising.
4. That has palpitation and excitement upon exertion.
5. That his appetite has changed and that he has frequent diarrhea.
6. That menstruations may be abnormal and that the sexual drive may be altered.
7. That the patient can tolerate cold temperatures better, or that he prefers cold to heat.
8. That there is emotional instability, since he cries more easily.

All or some of these elements, obtained only by the interrogation related to each system, help to round out the complete clinical picture of thyrotoxicosis.

REMOTE Anamnesis

In the remote anamnesis, and always following the order adopted in our clinical history, we must consider, first of all, the diagnostic interest of the personal history. This chapter includes the following: pathological history, which is collected from birth until the moment of the examination.

Knowledge of the diseases suffered by the patient in the course of their life often achieves great diagnostic value. Sometimes, the presence of a specific disease in the patient's history clarifies the cause of the disorders presented.

Other times, we will recognize in previous illnesses the preliminary stages of the current illness, which is nothing more than another episode in the course of the same chronically evolving process (pulmonary tuberculosis or syphilis, for example).

Thus we can determine the importance of acute joint rheumatism in heart diseases, especially in mitral valve lesions; that of streptococci in acute and chronic nephritis; that of typhoid fever in cholecystitis; that of mumps in orchitis and pancreatitis; that of measles, whooping cough and other anergic diseases in pulmonary tuberculosis; that of syphilis in aortitis, aneurysms and neurological lesions, etc.

In the personal history there are also other etiological factors of interest such as: lifestyle, diet, toxic habits, housing, reaction to medications, operations, immunizations, etc.

The way of life acquires great semiological importance, as it is responsible in some cases for the existing pathological process. Individuals with a dissipated life are exposed to venereal infections; such as heavy eaters, to obesity, diabetes, lithiasis and other nutritional conditions. In some cases, the way of life is closely linked to the profession and has been studied with it.

Knowledge of the type of diet used by the patient is of the greatest importance, and, thanks to it, we can reach the diagnosis of pathological processes as interesting as vitamin deficiencies. On other occasions, it is the water used as a drink that explains the existence of infections, such as typhoid fever, or certain endocrine diseases such as goitre, endemic in certain regions.

There is no need to overemphasize the diagnostic interest of discovering that the patient being examined has some of the well-known toxic habits (alcohol, coffee, tobacco) . In alcoholics, in addition to the very characteristic acute intoxication, the

alcoholic gastritis, polyneuritis, mental disorders, etc. In those who consume large amounts of coffee, such as heavy smokers, palpitations, tachycardia, decreased memory, gastritis and, especially in smokers, chronic pharyngitis and bronchitis and vascular spasms may be observed.

Likewise, it is important to know the environment that surrounds the patient, their home, to take into account overcrowding and the degree of hygiene in which they
It should also be studied in remote anamnesis, family pathological history or family or hereditary pathological history.

It is very useful and often necessary to do an interrogation in this section and establish what is called the person's family tree. In this aspect, fundamental data can be obtained to assess the genetic background of the patients. Let us remember that in this new century genetics will play a very important role.

family tree

The family tree or pedigree, although it is an essential instrument for the practice of human, medical and clinical genetics, is a way to obtain important information in a brief form, which allows us to appreciate numerous aspects of the family pathological history or family pathological history.

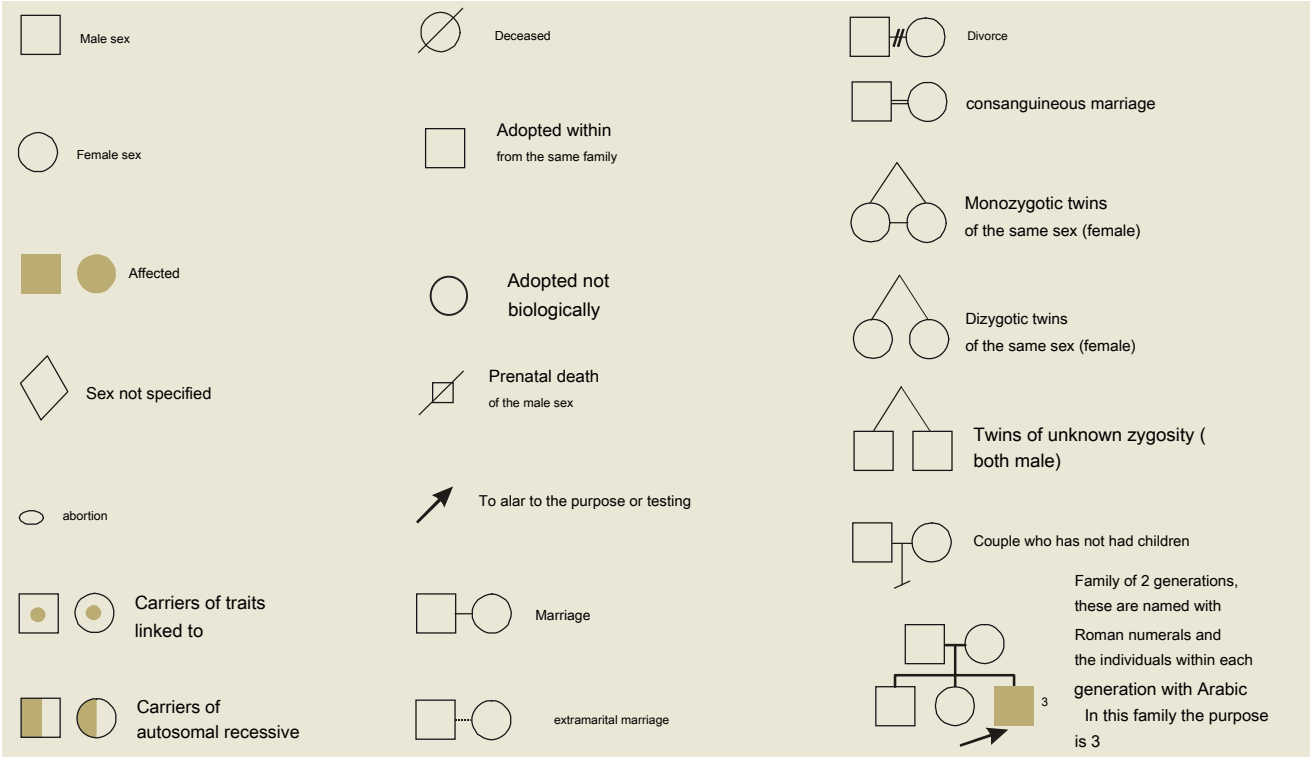
Its analysis can reveal the genetic commitment of the disease that afflicts the individual who requires medical attention. If we add to this the fact of the family repercussion of many of the conditions that are treated in the adult individual, such as apparently non-communicable chronic diseases, and the importance that family aggregation has in the etiological analysis in cases with these types of diseases, it will not seem far-fetched that in this Propedeutics text, the option of obtaining the family pathological history is offered using it.

Figure 23.1 provides the international symbols used to understand the language of the family tree.

Its completion depends on how the interrogation is carried out. The questions can begin with the inquiry about the number of siblings of the purpose (this is the name given to the original person who presents a mental or physical disorder and who serves as the basis for a hereditary or genetic study), by birth order and including the deceased, for whom the cause of death must be known and the birth order must be respected.

You should immediately ask if all the siblings are children of the same couple. If not, the divorce symbols are used, representing which siblings and half-siblings are. If the purpose

Fig. 23.1 International symbols used for the graphic representation of the family tree.


Male sex
Female sex
Affected
Sex not specified
abortion
Carriers of traits linked to
Carriers of autosomal recessive
Deceased
Adopted within from the same family
Adopted not biologically
Prenatal death of the male sex
To alar to the purpose or testing
Marriage
extramarital marriage
Divorce
consanguineous marriage
Monozygotic twins of the same sex (female)
Dizygotic twins of the same sex (female)
Twins of unknown zygosity (both male)
Couple who has not had children
Family of 2 generations, these are named with Roman numerals and the individuals within each generation with Arabic
In this family the purpose is 3
3

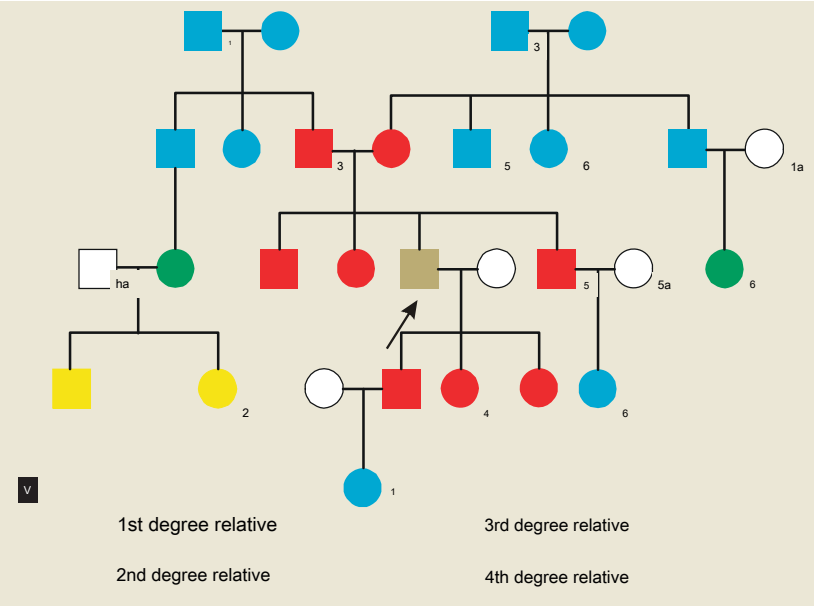


Fig. 23.2 Relationship of degrees of kinship with the purpose.

is of reproductive age, they inquire about their descendants, respecting in this case the order of birth of their children and asking again if they are children of a single marriage.

The symbols that define a generation must remain at the same level. The generations are identified with Roman numerals and each individual within each generation will be identified with Arabic numerals, so that when referring to the individual I-2, it is possible to know who is being referred to. For each couple you must always ask if there is consanguinity between them and use the corresponding symbol to identify them.

The degrees of kinship between the purpose and its relatives are represented in figure 23.2. First-degree relatives have a greater probability of genetic similarity between the gametes received. First degree relatives are the parents, siblings and children of the purpose; second degree are grandparents, grandchildren, uncles, nephews and half-siblings; the third degree includes first cousins and the fourth degree other relatives who are more distant from the purpose.

The number of generations will depend on the possibilities of the purpose of reporting the data or the possibility of questioning other family members. Data must be obtained from at least three generations of first-degree relatives.

example of data collection and its graphic representation in the family tree (fig 23 3)

Pablo, 50 years old and black, is admitted for a study of high blood pressure (HTN), which he has recently been diagnosed with. He has four siblings, two are boys and two are girls. Juan, the eldest, died at 56 years old due to a myocardial infarction (MI) and had been treated for HTN for three years; Ramón, 54 years old, appears to be in good health; Ana, 52 years old, is being checked with her family doctor, because on two occasions she has had high blood pressure with a minimum of 110 and Juana, 30 years old, suffers from bronchial asthma (BA). Juana is the daughter of another couple of Pablo's mother. Pablo's father is 80 years old and seems to be in good health, but his 78-year-old mother has been suffering from HTN for several years and has a history of overcoming an MI. Pablo did not know his father's brothers but he knows that his mother has four deceased male brothers, three living males and one living sister, all children of the same couple, although he does not remember information about his maternal grandparents.

Pablo has four children, all from the same marriage: Pablito, 26 years old, who has two three-year-old twin boys; Raúl, 24 years old, who has an 8-month-old daughter and Luis and Lucía, 18 and 15 years old respectively, who have no children.

When asking about the descendants of Pablo's brothers, it is known that Juan had two sons, the eldest died at the age of 30 from IM, leaving two sons who are currently 12 and 10 years old respectively; Juan's youngest son is 21 years old and has no children. Ramón does not have children. Ana has a 25-year-old daughter who also has no children and Juana has two sons, ages 5 and 3, who suffer from BA. Pablo's wife so far appears to be in good health and since she is not genetically related to Pablo, she has no interest in representing other members of his family. There is no reference to consanguinity in this story.

The analysis of this family tree allows us to identify the underlying genetic basis of the reason for admission of the

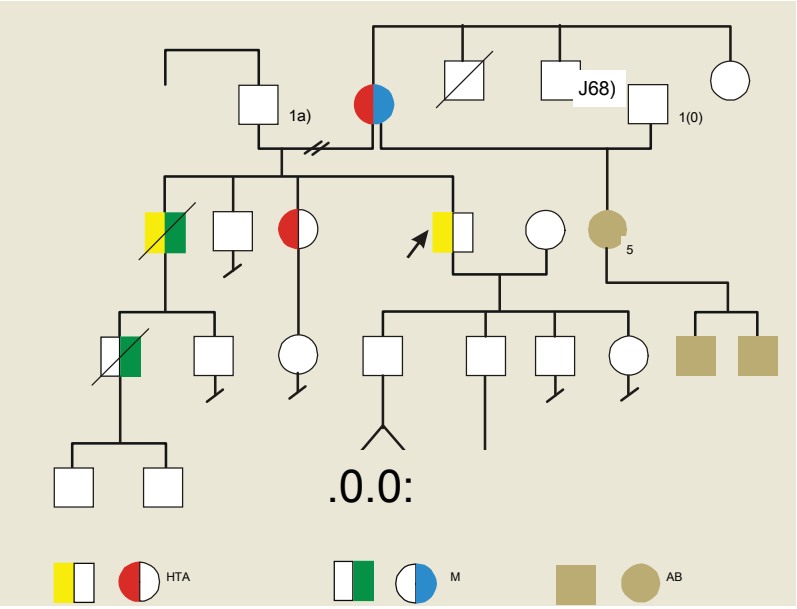


Fig. 23.3 Graphic representation of the example family tree.

I|-1: John; I-2: Ramón; II-3: Anna; I|-4: Pablo, the purpose. II|-4, II-5, III-6 and III-7 sons of Paul. III-1 and II-2: children of Juan. IV-1 and IV-2 grandchildren of Juan. I|-5: Joan; III-8 and I|-9: Juana's children.

purpose, and collect in a short space detailed information on four generations.

THE PHYSICAL EXAMINATION IN THE CLINICAL EXAMINATION

For years the physical examination has predominated in the clinical examination and medical capacity was judged by the art and science to practice it. The advent of more objective and reliable methods, such as imaging and others, has meant that it has been put aside.

However, we understand that it is essential that the doctor be trained in the essential aspects of these techniques and be able to obtain the essential physical signs anywhere to make a diagnosis, since he will not always have the opportunity to tell with more complex equipment, when it is far from hospital environments.

On the other hand, simple auscultation of the lung can accurately diagnose asthma, by listening to the typical wheezing rales, or acute pulmonary edema by identifying crepitant rales in the rising tide, which would not be easy to diagnose with other equipment, in addition to losing precious time in indicating the appropriate treatment.

Each of the four classic methods, described in Section I, acquires its greatest value in the different systems that make up the human body, as we will see throughout this text: let us point out as an example that in psychiatric conditions the interrogation is everything and, on the other hand, in neurological conditions the physical examination is essential.

Mod. 54-03

TO TO

Unit:

TO TO

TO TO

No.

FIRST SURNAME	MIDDLE SURNAME	NAMES	AGE: Completed years

Sex: Male ☐ Female ☐	Race: White O Yellow Or Mixed ☐ Black ☐ ☐	Birthdate Day	Month	Year	Place of birth:

Marital status: Single D Widower D Consensual union ☐ Married Or Divorced ☐ (Accompanied)	Economic classification Non-pensioner ☐ Pensioner ☐	Disease prevention certificate: Series:	Number:	does not have ☐ In process O

Occupation:	Branch of economic activity:	Occupation category: Employer D Self-employed Employee D Job. ☐ unpaid family member ☐

Address:

Location (city, town, etc.)	Neighborhood	Municipality	Province	Phone

Father's name: Alive ☐ Dead D	Mother's name: Live ☐ Dead D

AA A A

FIRST SURNAME	MIDDLE SURNAME	NAMES	PHONE

ADDRESS:

LOCALITY (city, town, etc.)	NEIGHBORHOOD	MUNICIPALITY	PROVINCE

SUBMITTED BY:

Were you injured at work? ☐ NO D	WORK CENTER	NAME: ADDRESS:

ENTRY DATE Day	Month	Year	HOUR: a.m. p.m. d	DISCHARGE DATE Day	Month	Year	HOUR: a.m. p.m.D	STAY: Days

HALL:	SERVICE OF:	ASSISTANCE DOCTOR:

Probable diagnosis		Doctor:
Diagnosis definitive		Doctor:

REASON FOR ADMISSION OR CONSULTATION:

HISTORY OF THE CURRENT ILLNESS:

FAMILY HISTORY:

PERSONAL BACKGROUND:

PHYSICAL EXAMINATION:

CONDUCT TO FOLLOW:

Mod. 54-05

T TO

REASON FOR ADMISSION: _____

HISTORY OF THE CURRENT ILLNESS: (description of the condition referring to its beginning, the chronological appearance of the symptoms, their evolution and therapy received)

PATIENT - First last name:	Second surname:	Names:	Medical record No. _____

Hall:	Service of:	Assisting doctor:

PERSONAL PATHOLOGICAL HISTORY:

Measles	☐	F. typhoid	☐	Asthma	☐	Blennorrhagia	☐	Encephalitis	☐		
Parotitis	☐	Influenza	☐	Urticaria	☐	Diabetes	☐	Poliomyelitis	☐		
Chickenpox	☐	Sinusitis	☐	Parasitism	☐	Rheumatism A.A.	☐	_____	☐		
Whooping cough	☐	Tonsillitis	☐	Dysentery	☐	High blood pressure	☐	_____	☐		
Diphtheria	☐	Bronchitis	☐	Malaria	☐	Jaundice	☐	_____	☐		
Rubella	☐	Bronchopneumonia	☐	Tuberculosis	☐	Appendicitis	☐	_____	☐		
Scarlet fever	☐	Allergy	☐	Syphilis	☐	Psychopathies	☐	_____	☐		

Reaction to medications (specify which ones): _____

Previous blood transfusion (specify if it has been done): _____

Trauma: _____

Operations: _____

Hereditary and family history: _____

Toxic habits:

Tobacco OR _____ AlcoholO _____

Coffee ☐ _____ Drugs OR _____

Lifestyle:

Daily work hours: _____

Daily hours of recreation _____

Feeding:

Good ☐

Fair Bad ☐

☐

Vaccinations received:

Anti-polio	☐	Tetanus toxoid	☐	B.C.G. _____	☐
Anti-typhic	☐	Quadruple	☐	_____	☐
Triple	☐	Anti-smallpox	☐	_____	☐

Disease prevention certificate:

Series:	Number:	
		does not have ☐
		In process ☐

Dwelling: _____

Floor:

Land

Mosaic

Cement

O00

Ceiling:

Guano ☐

Zinc ☐

Shingles ☐

Concrete ☐

Water:

In pipe inside the house — -Aqueduct ☐

From a cistern or well ☐

In pipe outside the home ☐

River Water ☐ Well ☐ Spring ☐

Sanitary services in the home:

Interior exclusive use - Lavatory / Latrine

Exterior exclusive use Lavatory / Latrine

Outdoor use of various No toilet Latrine / Toilet

or latrine ☐

DO0000

Bathroom or shower in the home:

Exclusive use ☐

Use of various ☐

No bath or shower ☐

Mod. 54-07

MEDICAL RECORD

	T AT TO
RESPIRATORY Dyspnea, cough, expectoration, hemoptysis, vomiting, cyanosis, pain, etc.	
CARDIOVASCULAR Pain, dyspnea, vertigo, palpitations, edema, headache, cough, asthenia, acroparesthesias, lipothymias, epistaxis, etc.	
DIGESTIVE Dysphagia, heartburn, heartburn, pain (characteristics), colic, nausea, vomiting, hematemesis, melena, enterorrhagia, food intolerance, aerogastria (eructation), aerocolia (flatus), diarrhea (characteristics), jaundice, hemorrhoids, constipation,	
GENITOURINARY Pain, hematuria, urethorrhagia, nocturia, dysuria, frequency, retention, incontinence, hemospermia, tumor, fistulas, stones, cloudy urine, urethral exudation, frigidity, impotence, eroticism, etc.	

PATIENT - FIRST LAST NAME: MIDDLE LAST NAME:	NAMES:	MEDICAL RECORD No. _____

HALL:	SERVICE OF:	ASSISTANCE PHYSICIAN:

GYNECOLOGICAL

last menstruation, menstrual formula, menarche, menopause, leucorrhoea, abortions, childbirth, menalgia, metromenorrhagia, amenorrhea, tumor, pain (characteristics), frigidity, etc.

HEMOLYMPHOPOIETIC

Lymph nodes, spleen, purple, capillary fragility, ecchymoses, petechiae, etc.

HIGHLY STRUNG

Headache, vomiting, head trauma, seizures (beginning and form): personality alterations. sensory, motor, senses (taste, smell, hearing, sight and touch), etc.

ENDOCRINE

Relating to: pituitary gland, thyroid, parathyroid, pancreas, testicles, ovaries or adrenals, etc.

OTHER DATA

Asthenia, anorexia, weight loss, fever, bleeding, osteoarticular system, etc.

CLINICAL HISTORY TAKEN BY (INTERNAL):	CLINICAL HISTORY SUPERVISED BY:

Mod. 54-06

MEDICAL RECORD

GENERAL Type, ambulation, gait, recumbency, facies, skin and mucous membranes, fanera, cellular tissue subcutaneous, current weight, height. SOMA, etc.	
REGIONAL AND BY SYSTEM	
RESPIRATORY Inspection, palpation, percussion, auscultation, cyanosis, dyspnea, indrawing, emphysema, effusion, shifte, ation, mediastinal	
CARDIOVASCULAR Pulse, blood pressure (max., min. and dif.), heart sounds, murmurs, rhythm, edema, hydrothorax, etc	

PATIENT - FIRST LAST NAME: SECOND LAST NAME:	NAMES:	MEDICAL RECORD No. _____
HALL:	SERVICE OF:	ASSISTANCE PHYSICIAN:

DIGESTIVE Mouth, tongue, throat, painful points: epigastric, pyloric, duodenal, pancreatic, common bile duct, vesicular, appendicular, Rovsing, Murphy, Blumberg, Meltzer, etc. maneuvers; hemorrhoids, rectal tract, etc. GENITOURINARY Abdomen, lumbar fossae, external genitalia, rectal examination, etc. GYNECOLOGICAL external genitalia, vaginal examination, bimanual palpation, speculum, etc. HEMOLYMPHOPOIETIC Lymph nodes, spleen, etc. HIGHLY STRUNG Motility, sensitivity, reflectivity, cranial nerves, etc.	
CLINICAL HISTORY TAKEN BY (INTERNAL):	CLINICAL HISTORY SUPERVISED BY:

Mod. 54-19

MEDICAL RECORD

TO T.A.	

PATIENT - FIRST LAST NAME: SECOND LAST NAME:	NAMES:	MEDICAL RECORD No. _____

HALL:	SERVICE OF:	ASSISTANCE PHYSICIAN:

T TO TO TO T.A.	

Mod. 54-03

T TO TO

Medical record

Unit:

TO

Reason for admission:

Syndromic summary:

Summary of physical
examination (data of

Treatment:

Complete OR Incomplete ☐ Not practiced ☐

Operations:

Evolution:

Satisfactory ☐ Not satisfactory D Complications

Definitive diagnosis upon
discharge:

Other diagnoses:

Result:

Cured ☐ Equal ☐ Unspecified ☐

Improved D Worsened ☐ Dead

iBefore 48 hours ing. ☐

After 48 hours Autopsy No ☐

OBSERVATIONS:

			Hour:
Day	Month	Year a.m. D p.m. ☐	

Signature of the discharging doctor

PATIENT - First last name:	Second surname:	Name:	Medical record No.
Hall:	Service of:	Assisting doctor:	

24

GENERAL INSPECTION

Inspection is one of the four classic clinical examination procedures. In it we use the sense of sight, and we distinguish a direct or immediate inspection, which is one in which we use only this sense, and another, instrumental mediatao, in which we use instruments or devices.

Semiotechnics

Once good illumination is obtained, we must undress the patient, respecting, however, the mandates of modesty, especially if it is a woman, in whom we will only do so partially and with the greatest delicacy. The patient will stand, sit or lie down, and the observer will stand in front of him, with his back to the light, and will proceed to collect the inspection data in strict order. For more details of the exploration, review Chapter 3, in Section I.

EU DATA IS COLLECTED BY THE GENERAL INSPECTION

As soon as we come into contact with the patient we observe whether he or she walks normally or with difficulty, or if he or she is forced, to a greater or lesser degree, to remain in bed.

The fact that the patient comes to us on his feet generally entails the possibility that he is not suffering from a serious condition, while when we are requested at his bedside, it is almost always a case of serious or acute illnesses.

This statement has only a very relative value, since various conditions - individual, social - can falsify it.

We frequently observe in our hospital services, patients with serious conditions who arrive walking. It is the greater resistance and energy of the sick individual that allows him to continue standing, even with significant conditions. Other times, there are morbid processes of extraordinary severity that, even at a time close to the patient's death, are compatible with walking. We have had the opportunity to observe patients with advanced carcinomas frequently attend consultations until very shortly before their death.

Some diseases require bed rest, such as those accompanied by intense pain, muscular impotence or extensive paralysis, very high fever, vertigo, intense dyspnea or marked prostration, such as

to be seen in certain abdominal, osteoarticular, nervous conditions or acute lung or heart infections.

In certain infectious diseases - typhoid fever, above all - patients are observed who, despite exhibiting its symptoms (fever, headache, digestive disorders, for example) continue with their activities and go to the doctor's office on their own. These are the so-called ambulatory forms, which can not only be found in typhoid fever, but also in pneumonia and other conditions.

Another piece of data of greater importance that we collect through the general inspection is the attitude of the patient, whether in bed or in the standing position.

Patient's attitude in bed

When we examine a patient lying down we must carefully observe the position he adopts in bed, that is, what the decubitus position is (from the Latin decumbo:
lie down) that presents.

We will distinguish a dorsal or supine decubitus, a ventral or prone decubitus and a left or right lateral decubitus, depending on whether the patient is lying on his back, on his belly or on one of his sides.
Some authors also consider as recumbency the semi-sitting or sitting position that certain patients are forced to adopt, especially those who suffer from intense dyspnea.

The recumbent position that a patient presents may be the one adopted by his own will and strength or it may be the one in which external forces, especially gravity, place him, because he is inert, lacking strength or unconscious. In the first case, we say that it is an active decubitus; in the second, a passive decubitus position.

Passive decubitus

Passive recumbency or dorsal inertia, as it is also called, is a passive attitude. The patient usually lies on his back, with a tendency to slide toward the foot of the bed, or elsewhere. It maintains the position in which it is placed on the bed, as long as it does not counteract the action of gravity.

Passive decubitus is usually found in cases where the patient has lost consciousness or is extremely weakened, without strength. It manifests great muscle weakness and mental apathy.

We observe passive recumbency in various morbid states, almost all of them serious. In diseases of the nervous system, especially those that lead to a state of coma or semi-coma (cerebral hemorrhage, for example); in acute infectious diseases, especially in the most frequent clinical form of typhoid fever, from which it takes its name (tufos: stupor) due to the stuporous state it produces; in the co-

period, with its marked peripheral cooling, sweating, tachycardia, arterial hypotension, etc., which always characterizes serious processes, such as large hemorrhages, serious abdominal conditions, trauma or operations (traumatic or surgical shock).

Passive recumbency can also be observed when there are extensive paralysis or marked muscle hypotonia, as in certain cases of muscle atrophy, tabes, or congenital myotonia or Oppenheim's disease.

active decubitus

Active recumbency is one in which the patient participates by his or her own will and strength; It can be indifferent or forced depending on whether or not it is modified at will, without inconvenience or annoyance.

The indifferent active recumbency offers little semiological interest. This is not the case with forced or forced active recumbency, which is important for the diagnostic guidance it often provides.

Forced active decubitus is found mainly in those diseases that are accompanied by dyspnea, pain, paralysis, muscle contractures or tendon retractions, joint disorders, etc.

Among them we have:

- Orthopnea position.
- Pillow sign.
- Mohammedan prayer.
- Forced lateral decubitus.
- Dorsal recumbency.
- Prone position.

Orthopnea position

Forced standing or sitting position adopted by the patient suffering from intense dyspnea or orthopnea (from the Greek orthós: straight; pnoiá: breathing).

Concept and semiography

In orthopnea, the patient, suffering from intense respiratory distress, is sitting or semi-sitting, resting on several pillows to keep the trunk erect, the only position in which he is relieved and can breathe more easily. In extreme cases, the patient leans forward, resting his hands or elbows on a resistant plane; Sometimes he leaves the bed to lie on a window, on a table or in a chair with his body reclined forward (fig. 24.1).

Semiogenesis or pathophysiology

This attitude undoubtedly tends to make breathing easier, since with the trunk erect, the movement of the diaphragm is helped by the descent of the abdominal viscera.

Fig. 24.1 Orthopnea.

and, when you rest your arms on a fixed plane, these and the neck can act on the auxiliary respiratory muscles (sternocleidomastoids, scalenes, pectorals, trapezius, etc.), which ordinarily have their fixed insertion in the thorax and their movable insertion in the upper limbs or the head. In this attitude, the contraction of these muscles determines a lifting of the clavicle and the first ribs, which contribute to greater chest expansion.

In the orthopnea position, vital capacity increases, pulmonary ventilation is easier, and cardiac output and pulmonary congestion decrease, factors that favor gas exchanges. Let's study them below:

Increase in vital capacity. Vital capacity is generally greater in the sitting position than in the supine position, although Hamilton and Morgan (1931) found that the thorax has greater amplitude in the latter position. They attribute the lower vital capacity in the supine position to the fact that the lungs contain a greater amount of blood and, in addition, to the fact that the diaphragm is higher.

Better lung ventilation. The respiratory muscles and the diaphragm perform their function more easily, since the abdominal viscera tend to descend due to the action of gravity. Other authors have verified that the respiratory rate increases in these patients when they adopt the supine position.

Decrease in cardiac output and pulmonary congestion. Cardiac output is lower in a sitting position and, therefore, so is the work of the heart; It reduces blood flow to the right heart as there is venous stasis in the lower limbs, which reduces pulmonary congestion and thus the stimuli that act on the respiratory center.

Semidiagnosis

Orthopnea occurs when there is left ventricular failure, but does not appear in right ventricular failure. It can be observed, therefore, that some patients with exclusive or predominant failure of the right ventricle (pulmonic stenosis, tricuspid regurgitation, etc.) can lie horizontally without experiencing greater dyspnea. It is also seen in severe lung conditions and acute asthma attacks.

cushion sign

In some cases of large pericardial effusions or hearts considerably enlarged in volume, even more forced positions are observed. Some patients place a pillow on their thighs and, remaining seated, lie down on it, adopting a very peculiar attitude that has been described as the "pillow sign" (fig. 24.2).

Mohammedan prayer

Other times, the greater intensity of these processes forces the patient to adopt the "Mohammedan" or genupectoral prayer position, in which the patient is kneeling on the bed, inclining his trunk strongly towards the plane of the bed, leaning on it with his hands or his elbows. These attitudes assumed instinctively by the patients, by projecting the heart with its pericardial envelope forward, contribute to the decompression of the lung (rejected backwards by the heart). hypertrophic or pericardial effusion), thereby facilitating breathing.

Fig. 24.2 Cushion sign.

Forced lateral recumbency

Forced or forced lateral decubitus is mainly observed in conditions of the respiratory system and is conditioned by pain, dyspnea and cough with expectoration, present. Thus, patients with an intense pain in their side due to pneumonia or pleuritis generally lie on their healthy side, so as not to increase the pain. Subsequently, in the case of pleural inflammation, if it produces effusion, it is observed that, as the fluid collection increases and the pain decreases, the patient tends to lie down on the affected side, a position adopted when the effusion reaches a certain volume, since this allows the healthy lung to breathe more freely and compensates for the respiratory deficit resulting from atelectasis, determined by the fluid collection on the diseased side. A similar attitude is usually observed, for the same reasons, in pneumothorax, pyopneumothorax, hydropneumothorax, large lung tumors, etc.

Forced lateral decubitus or, better expressed, preferred lateral decubitus, can be observed in cases where there are suppurating cavities in the lung (pulmonary caverns, bronchiectasis). In these cases, the patient usually lies on the same side as the suppurating cavity. This way you avoid being constantly woken up by the cough caused by the passage of purulent secretion from the cavity into the bronchi. During the night and due to the action of gravity, facilitated by the adopted lateral decubitus position, the pus accumulates in the cavern or dilated bronchus, to be expelled in gulps when getting up. The location of the suppurating cavity determines, as is easily understood, the forced recumbency adopted. This is how some patients with cavities in the lower lung lobe look, preferring a sitting or semi-sitting position, and others, with anterior cavities, who choose the ventral decubitus position.

Forced lateral decubitus can also be observed in heart patients, who, when disturbed by the heartbeat in cardiovascular erethism, or in heart hypertrophy, adopt the right lateral decubitus. In cases of large cardiac dilation, patients choose the left one to facilitate the work of the right lung and relieve the dyspnea that sometimes occurs due to lung compression.

Dorsal recumbency

It is adopted by patients affected by acute abdominal processes (appendicitis, peritonitis, cholecystitis), and is frequently accompanied by flexion of the lower limbs, or one of them, the right, for example, as seen in appendicular inflammation.

Prone position

It is often adopted by those suffering from abdominal cramps, such as saturnine cramps, which are seen in lead poisoning, and which are appreciably relieved in this position. Likewise, some cases of epigastralgia due to an ulcer of the posterior wall of the stomach occur in the prone position, and cases that suffer from certain injuries to the spinal column, for example, tuberculosis (Pott's disease).

The following special attitudes can also be observed, mainly in diseases of the nervous system:

Opisthotonos

(From the Greek opisthen, backwards; tones: tension.) It is a rare attitude, in which the body rests on the head and heels, arching the trunk upwards; The back is concave, towards the plane of the bed, in such a way that you can pass your hand between them. It is seen in strychnine poisoning, tetanus, convulsions of rabies, hysteria, epilepsy and, to a lesser extent, meningitis (Fig. 24.3).

Emprostotonos

In emprostótonos (from the Greek émprosthe: forward; tones: tension), the position adopted by the patient is the reverse of opisthotonos. The body bent forward rests on the forehead and feet, with the face downwards. It is much rarer than opisthotonos and is seen in strychnine poisoning and tetanus; In the latter it gives rise to what is called ball tetanus.

Pleurototonos

In this attitude the curvature of the body is lateral, arching on one side, due to unilateral contracture.
It is seen, very rarely, in tetanus or in some conditions of the spine or pleura.

Fig. 24.3 Opisthotonos.

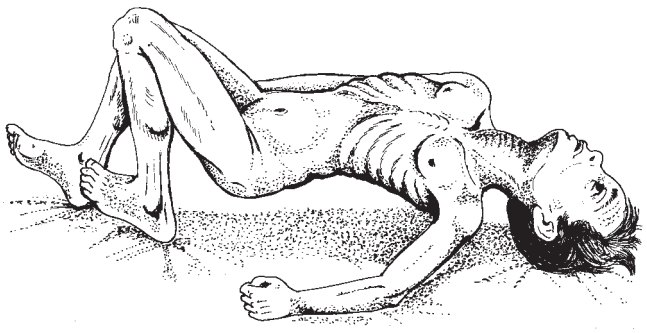


Fig. 24.4 Shotgun trigger position.

Etymologically, the term comes from the Greek pleurotheu: sideways; tones: tension.

Shotgun trigger position

In tuberculous meningitis, as a consequence of the muscle contracture that causes irritation of the cerebral cortex, the so-called shotgun trigger position is sometimes observed. The patient is in a lateral decubitus position, with slight extension of the neck and marked adduction and flexion of the thighs on the abdomen and the legs on the thigh, his silhouette simulating a shotgun trigger, from which he takes the name with which the French authors have baptized it. (fig. 24.4).

Attitude during standing position

It should always be observed, since in many cases it is very characteristic and of great diagnostic value.

The attitudes or ways of standing are divided into:

Somatoconstitutional, or normal, or physiological attitudes. They are those that reflect the "constitutional type" of the individual in its somato-temperamental aspect.

Pathological attitudes. They are those that are determined by the functional consequences that some diseases can produce.

Somatoconstitutional attitudes

For the semiological appreciation of these attitudes, as well as for pathological attitudes, the individual must be observed from its various angles: frontalis, lateralis and posteriori; analyze and consider the static relationship between the head, trunk, upper and lower limbs; observe its line of verticality, its angles and curves; appreciate the sensation of rigidity or flaccidity that the various segments present, depending on the resistance they oppose to gravity, that is, the static synergy that the various segments of the body have among themselves. It is also advisable to observe the individual's way of sitting and standing, as well as the energy, rhythm, mo and the manner of his gestures and movements (see " Attitude" in Section).

Somatoconstitutional attitudes are a result of the diverse tone and posture that the ligamentous-muscular system can present in a normal state, which, in turn, depends on the autotone of its tissues, regulated and controlled by the nervous, vegetative and relational system. It is thus explained that these attitudes represent the somatic and temperamental modality of the individual.

Somatoconstitutional attitudes are of two types:

Aesthetic type

Which is one that presents a good tone of the ligamentous-muscular system, which is manifested upon inspection by an attitude that gives the impression of poise, ductility and energy at the same time; for example: the attitude of the man at arms and that of the athlete.

asthenic type

Which is one in which the ligamentous-muscular tone suffers from a constitutional deficit of regulation, which is why this type of attitude gives an impression of abandonment and flaccidity in its various body segments. Thus, the head is not supported with aplomb, the arms hang pendulous to the sides of the trunk, the shoulders appear a little loaded forward, and the legs present (as well as the trunk) a slight bend: for example: the attitude of the sedentary man.

Pathological attitudes

We will point out that they are almost always conditioned by the existence of: pain, bone and/or joint, muscle or nervous system conditions. Without going into more detail in the corresponding chapters, let us point out just a few examples.

In patients who suffer from painful gastric or intestinal diseases, when they can stand, a characteristic attitude is observed, consisting of bending the trunk forward, supporting the belly with the hands. If the pain is experienced in the lumbar region, or in the hip, the body leans towards that side, flexing the corresponding lower limb and lowering the shoulder on the same side, which makes the subject assume a characteristic inclined position. In chest pain (pneumonia, pleurisy, intercostal neuralgia), the patient presents with the trunk flexed on the affected side, where he frequently places his hands on it to try to relieve the pain by thereby reducing respiratory chest expansion.

The attitudes that many central nervous system patients assume when standing are also very characteristic. Among them we have the special attitude

Fig. 24.5 Attitude in hemiplegia.

of hemiplegia, with the shoulder dropped and the upper limb in flexion (fingers flexed on the hand, hand on the forearm and forearm on the arm) and in adduction (close to the trunk), and the lower limb in extension, which allows the characteristic gait of these patients (fig. 24.5). In Parkinson's disease, or shaking paralysis, the attitude of the patient is typical, who appears fixed, like a soldier, slightly flexing his trunk forward, as if in a saluting attitude, with a characteristic, stuporous and almost immobile facies (fig. 24.6). It is interesting to mention the so-called tenor attitude, which is observed in certain primitive myopathies, in which the patient presents marked depression.

Fig. 24.6 Attitude in Parkinson's disease.

Fig. 24.7 Attitude in tenor position.

lumbar, in the shape of a saddle, extension of the trunk backwards, which, with the great separation of the feet, gives the sensation of a singer who is going to emit a high note (fig. 24.7).

Brand

It is another of the important data that we obtain, from the first moment, in the general inspection of the patient. Of great diagnostic value, especially in diseases of the nervous system, it will be studied in detail in the chapter corresponding to this system.

We will point out the most salient types here.

Simple observation of the behavior of different people always shows something peculiar, individual, in each case. It will be understood that, in a pathological state, in certain nervous diseases, certain types of gait are recognized as a characteristic. It should be clarified that when studying pathological gait, not only the way of walking is observed, but also the position of the body, the movement of the arms and the attitude of the head (fig. 24.8).

In the ataxic (heel-heel) gait, the patient suddenly projects his foot forward and then abruptly places his heel on the ground. This gait is observed in the tabes dorsalis.

Another characteristic gait is the polyneuritic or steppe gait, observed in polyneuritis or polyneuroradiculitis of the lower limbs of different etiologies (diabetic, alcoholic, arsenical). In it you can see that the patient raises his leg widely, leaving the foot as if hanging.

The scything or hemiplegic gait of spasmodic hemiplegia is one in which the patient moves the leg.

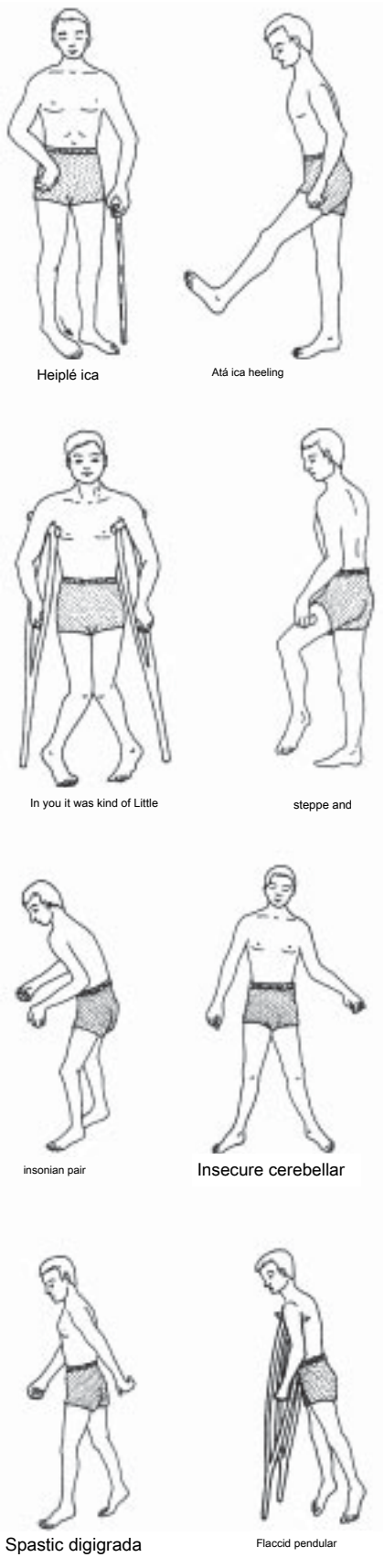


Fig. 24.8 Most frequent types of gait.

It affects by tracing an arc of external convexity that recalls the movement of the scythe.

Hesitating, asynergic or cerebellar gait is also worth mentioning. Here the patient walks in a zigzag (drunk gait), he also has deviation towards one side or the other of the destination point (lateropulsion), he also has hesitations. It is seen in cerebellar syndrome.

external sphere

The three large groups, according to the body conformation of the subject, their external habit or biotype, were already described in Section I of this work (see Chapter 3), and we will carry out a detailed study of the important problems raised by these considerations later. Here we will deal with the general inspection of the patient, the data that refer to the alterations in his external habit and constitution.

Growth and size or height of the patient

Growth is rapid in the first years of life - especially in the first year - and gradually decreases until puberty, when a growth spurt occurs until reaching, almost completely, the subject's final size. The height may be increased or decreased, which constitutes gigantism or dwarfism, respectively. Gigantism, height greater than 2 m, is generally due to disorders of the internal secretion glands, especially the anterior lobe of the pituitary gland (Fig. 24.9).

Dwarfism, height less than 1.20 m, may be due to endocrine disorders (mainly thyroid or pituitary), or to lack of growth of the extremities due to damage to the epiphyseal cartilages. This is the case of achondroplasia (from the Greek a: privative; chondros: cartilage; plasia: formation). Achondroplastic dwarfs have very large heads and trunks in relation to the shortness of their limbs (fig. 24.10).

Every patient must be weighed and their weight compared to the generally accepted standard for a person of the same sex, age and height (see tables in Section I, Chapter 3).

Weight that does not deviate from the standard, neither more nor less than 10% of the normal figure, can be accepted as normal. When the subject's weight is above the limits indicated as normal, we speak of obesity; if it is below, thinness.

Obesity is due to the storage of fat that results from excessive nutrition (exogenous factor) for the needs of a normal metabolism (endogenous factor).
Obesity will be exogenous or endogenous when it is mainly due to one of the aforementioned factors. Exogenous is mainly related to excessive feeding and

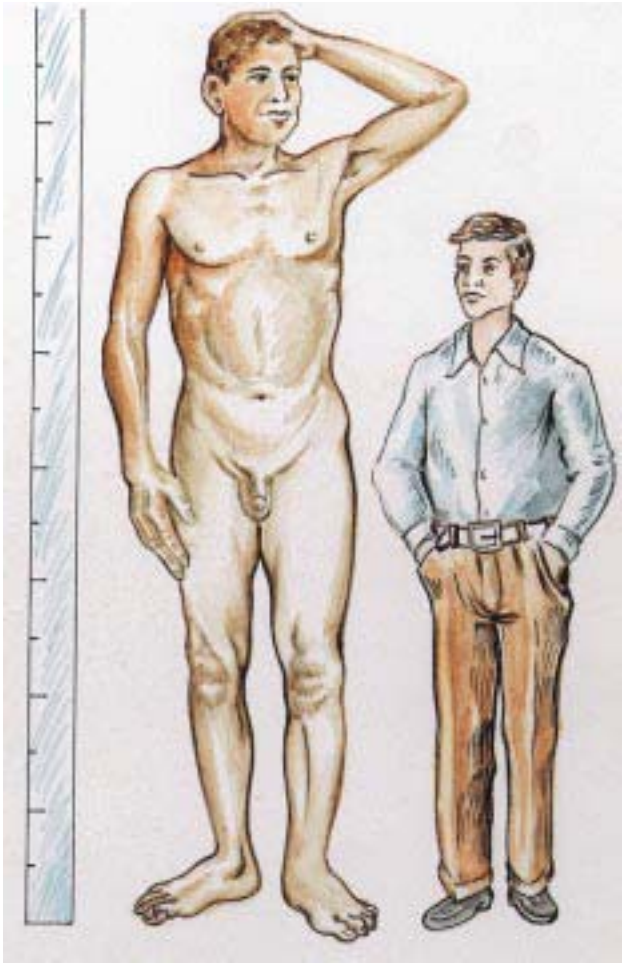


Fig. 24.9 Gigantism.

Fig. 24.10 Achondroplastic dwarf.

sedentary lifestyle; endogenous, with a decrease in basal metabolism, and is fundamentally determined by disorders of the internal secretion glands, such as the thyroid, pituitary, adrenals and gonads.

Thinness can also be exogenous (hyponutrition, anorexia), and endogenous (due to increased basal metabolism, generally caused by thyroid hyperfunction).

Weight loss can be observed in pulmonary tuberculosis, in neoplasms, especially those of the stomach and pancreas, in diabetes mellitus, in hyperthyroidism, in pituitary insufficiency (Simonds' disease), etc.

Cachexia (from the Greek kakos: bad; axis: state) is a special type of severe malnutrition with weight loss in which the proportional relationship between malnutrition and weight loss is broken (Fig. 24.11). It is observed in anorexia nervosa, in Simonds cachexia, in the terminal period of long-term illnesses.

Facies

The facial or physiognomic expression is extraordinarily rich in valuable data for diagnosis. The importance of these data and the almost infinite number of facies described would make their study unapproachable, if we did not limit ourselves to studying the most important pathological facies, classically described.

In reality, the study of facies is eminently objective, so we will limit ourselves here to recording their most salient characteristics and we will complete their study as we find them in practice. We will order their study by systems, reserving for last the description of the most interesting facies due to general processes.

Among the most interesting facies found in diseases of the respiratory system, we will choose two of them for study: the adenoid facies and the pneumonic facies.

Adenoid facies

It is very characteristic. It generally occurs in children who suffer from adenoid vegetations (from which it takes its name) or other causes of obstruction of the nasopharynx. As a consequence of the difficulty in nasal breathing that these processes determine, the subjects who suffer from it generally have a constantly open mouth, with the lower jaw drooping and protruding, a thin nose, with its poorly developed openings, and an unintelligent expression of the face (even when a well-developed intelligence is preserved) that completes in a very expressive way the character of this facies (fig. 24.12).

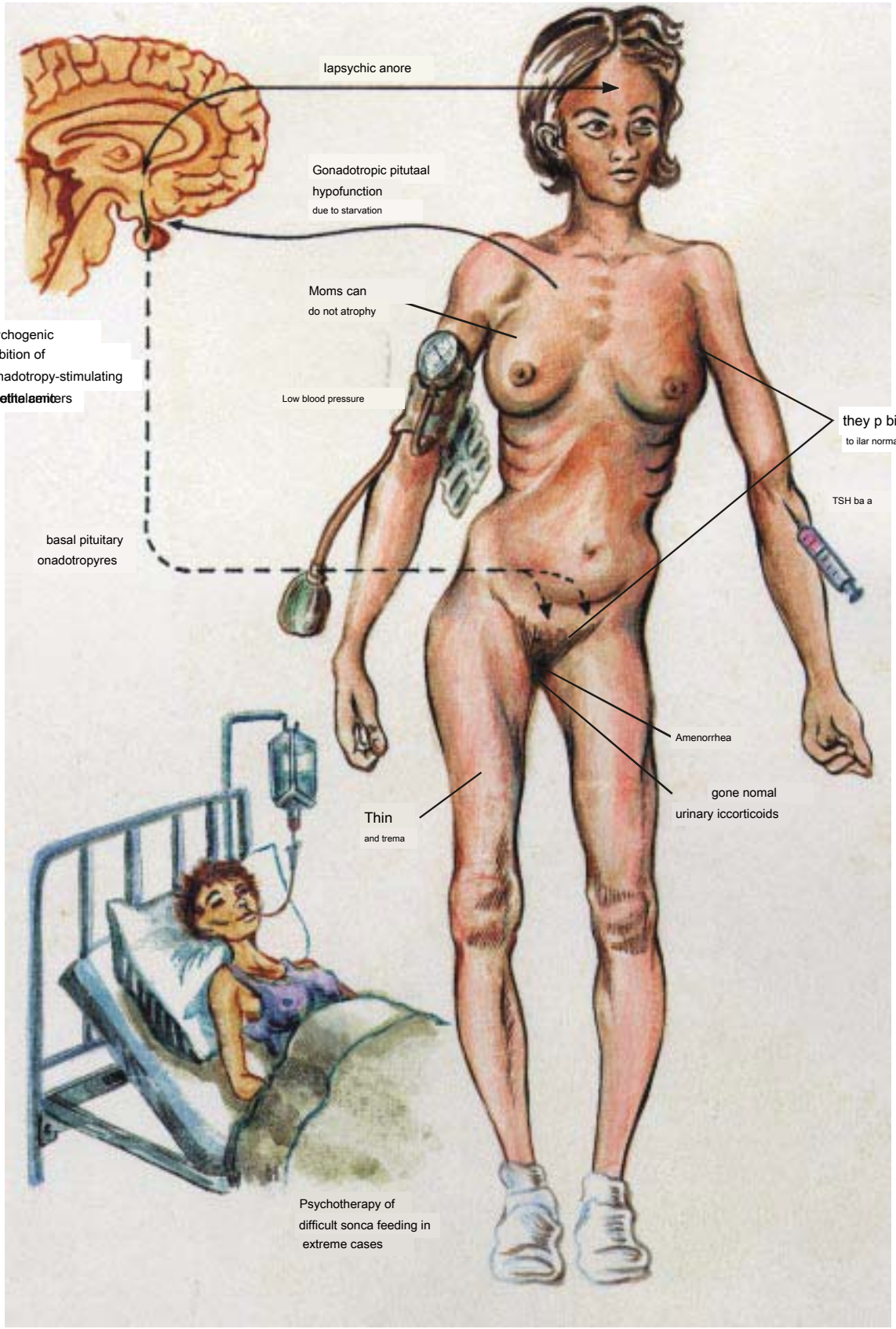


Fig. 24.11 Cachexia: anorexia nervosa.

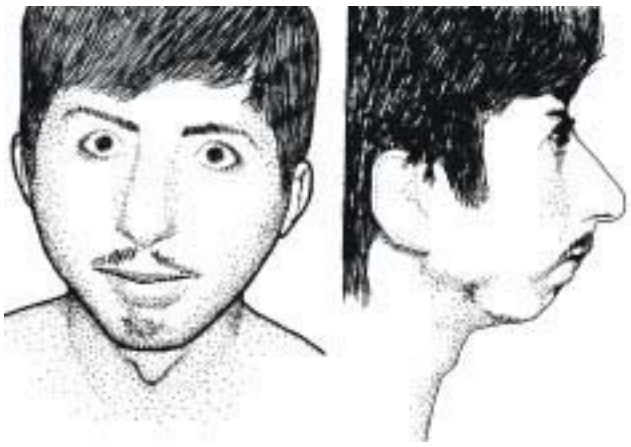


Fig. 24.12 Adenoid facies: a, front; b, profile.

Fig. 24.13 Renal facies: a, mild; b, extreme.

Pneumonic facies

In her we observed her bright eyes, somewhat anxious due to dyspnea, and a very visible redness on one of her cheeks. The flaring of the nose produced by dyspnea and the appearance of herpes vesicles around the corner of the mouth completes this facies.

In the circulatory system, the aortic facies and the anxious facies of asystole are interesting to study.

Aortic facies

It is observed in subjects with insufficiency of the aortic sigmoid valves. There is paleness of the face, although sometimes a slight cyanotic tint can be perceived; wide arterial beats in the temporal arteries and especially in those of the neck; Sometimes a special movement of the head is observed as if they were making constant affirmative gestures with it (Musset sign).

Anxious facies of asystole

The expression of anxiety that gives it its name stands out, the pronounced cyanotic color of the face, which is edematous in many cases, and the constant flaring of the nose, expressive of the intense dyspnea that characterizes these patients.

Among the facies of kidney diseases, the so-called renal or edematous facies deserves a special study.

Renal or edematous facies

It is characterized by edema of the face, especially the eyelids, and paleness of the skin, secondary to the edema itself. The edematous infiltration rounds the face, which, with the narrowing of the palpebral opening that determines the edema of the eyelids, gives the whole an appearance very similar to that of the dolls found in many Chinese drawings, which is why this facies is also called Chinese doll facies. It is observed in nephritis that gives rise to edema and in nephrosis (Fig. 24.13).

In diseases of the digestive system we will study the peritoneal facies and the hepatic facies.

Peritoneal or peritonitic facies

Also called Hippocratic facies. It is observed in serious abdominal processes (septic peritonitis, gastric or duodenal ulcer perforations, intestinal perforations, strangulated hernias, etc.) and in the agonic stage of other diseases. It is characterized by the anxious expression and earthy paleness of the face with sunken eyes and cheeks, especially the first ones, which seem to get lost in the depths of the orbits, bordered by violet circles under the eyes. The sinking of the soft parts highlights the nose and the bone formations of the face, the former appearing as elongated, thinned and sharp, which, with the marked protrusion of the cheekbones, gives a peculiar and impressive appearance to the physiognomy. This, together with the vague, torpid look of these patients, faithfully reflects the seriousness of the process and the imminence of its fatal outcome. The outward displacement of the earlobe due to local contraction completes the set of facies characteristics with such a serious prognosis.

Hepatic facies

It is observed in those who suffer from liver diseases, especially chronic march, and in liver failure. It is essentially characterized by a yellow or straw tint of the skin, in which small spider-shaped vessels called spider veins or arachnoid telangiectasias can be observed. Sometimes dark pigment spots are observed, especially on the cheeks and forehead.

But where numerous and very interesting facies are found is in diseases of the internal secretion glands, that is, in endocrinopathies. Among them we will study the acromegalic, cretinoid, myxedema and baseowian facies.

Acromegalic facies

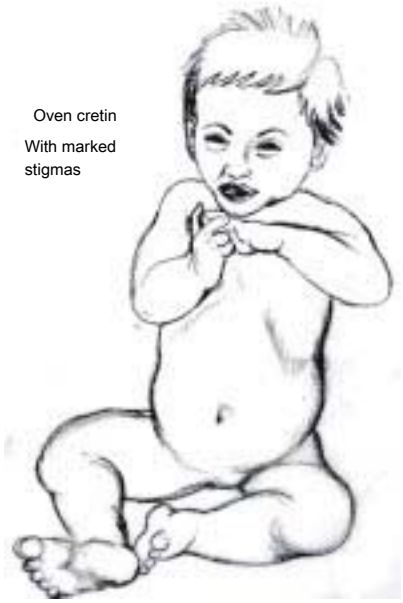
It is characterized by the considerable development of the skeleton of the skull and face, and the thickening of the cranial vault; notable increase in size of the external occipital protuberance and frontal protuberances; great development of the cheekbones and zygomatic arches and, especially, of the lower jaw, which becomes prognathic. The nose increases in size in a very characteristic way, developing, especially in width. There is also hypertrophy of the tongue with greater prominence of its papillae and spacing or separation of the teeth that is due to abnormal growth of the lower jaw (fig. 24.14). These bone growth disorders are not limited to the skeleton of the face and skull, but also affect the other bones, except the long ones, which are very little altered. In these cases, marked growth of the hands and feet is observed, which awakens the patient's attention as he is forced to progressively increase the number of shoes or gloves he uses. This process is related to the existence of a hypersecretion of growth hormone, produced by the eosinophilic cells of the anterior lobe of the pituitary gland.

Cretinoid facies

It occurs in endemic cretinism and in rare cases of sporadic cretinism. In both, it is essentially a state of hypothyroidism that begins during fetal life.

It is characterized by a wide face, with thick lips and the mouth, generally open, allowing the enlarged tongue to peek out. The nose is wide and flattened, the hair is sparse and fragile, and the skin is dry and thickened, with a brownish tinge. These patients have an expression

Fig. 24.14 Acromegalic facies.



Oven cretin
With marked stigmas

Fig. 24.15 Cretinoid facies.

stupid and very limited intelligence, deaf-muteness or muteness is frequently observed among them (fig. 24.15).

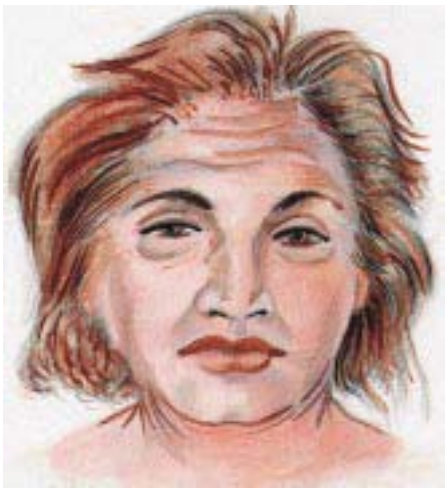
Myxedema facies

In it the general expression is one of apathy and stupor; the skin is rough, dry and thick; the cheeks often cyanotic and the eyelids bloated. The large and slightly turned outward lips reveal the mucosa of the mouth; The nose is wide, the ears are thick, the hair is sparse and tends to fall out, like the eyebrows, which generally lack the tail (eyebrow tail sign). It is observed, above all, in myxedema caused by a deficiency in thyroid secretion (hypothyroidism) (fig. 24.16).

Facies based on iana or hyperthyroid

It is a very characteristic facies and of great diagnostic value. It is observed in cases of exophthalmic goiter,

Fig. 24.16 Myxedema facies.



where there is thyrotoxicosis or hyperthyroidism, with an increase in the volume of the thyroid gland (goiter). In these cases, the patient's appearance seems to correspond to that of a person who had suffered a severe scare. Added to the frightened physiognomic expression, as if of terror, are the protruding, bulging eyes (exophthalmia), very open, with a large opening of the palpebral slit, and the almost complete absence of blinking. The absence or decrease of blinking has been described as Stellwag's sign. The large eyelid opening is known as Dalrymple's sign.
The Von Graefe sign can also be observed here, which consists of the fact that when looking downwards, the upper eyelid is set back, in such a way that the sclera becomes visible above the iris (fig. 24. 17).

In disorders of the nervous system we will also find very interesting aspects. We will refer only to the facies of facial palsy and Laparkinsonian paralysis.

Facies of facial paralysis

It is characterized by the deviation of the mouth towards the healthy side in the initial stages, with lowering of the lip commissure and absence of facial folds in the paralyzed half of the face. In peripheral facial paralysis, the impossibility of closing the eye on the diseased side is also observed, due to lack of descent of the upper eyelid, which determines a special appearance of the eye that is reminiscent of that of statues.

This is known as Bell's sign, which is useful as a differential diagnostic element with facial palsy of central origin, in which this sign is not observed (fig. 24.18).

Parkinsonian facies

It is very characteristic. The immobility of the face, and the lack of any mimic expression, gives it the appearance of a mask; the physiognomic expression of the subject is as ad-

Fig. 24.17 Ianian or hyperthyroid based facies.

Fig. 24.18 Facies of peripheral facial paralysis.

gaze or fright. The eyes often appear intelligently expressive, as if they wanted to compensate for the inexpression of the rest of the face (fig. 24.19).

Other facies that we can find in different conditions are:

Mediastinal facies

The purplish discoloration of the face that appears bloated - frankly edematous -, dyspnea and engorgement of the veins secondary to compression of the superior vena cava in the mediastinum stand out. In these cases, cyanosis and edema usually extend to the head, neck, upper extremities, and upper chest, which is described as cyanosis or slave edema.

Painful facies

The patient's expression of suffering and the increase in the transverse folds of the forehead and contraction of the muscles of the face are very characteristic of this facies.

Fig. 24.19 Parkinsonian facies.

Feverish facies

In general processes accompanied by fever, the existence of this facies can be seen, with its bright look, redness of the cheekbones and paleness of the rest of the skin, possibly determined by the existence of a certain degree of sympathetic excitement.

Some infectious diseases present very interesting features. We will study among them, the tetanic facies, and the typhoid or stuporous facies.

Tetanic facies

The existence of a permanent laughing expression in the mouth (sardonic laughter) stands out, with contracture of the muscles of the face, and, frequently, of the masseter muscles, which prevents the opening of the mouth (lockjaw) (fig. 24.20). The picture is completed by convulsions, fundamentally tonic, which frequently lead to the adoption of special attitudes, opisthotonos for example.

Typhoid or stuporous facies

Characteristic of typhoid fever and other serious toxic-infectious processes, what stands out above all is the expression of drowsiness or drowsiness and great exhaustion of the patient, who remains prostrate, with his eyes closed or half-open and his lips dry and covered with small dark concretions that are called fuliginosities.

Constitution

Concept and importance of the constitution

The extraordinary importance that studies on the individual constitution acquired at the time, developed to the point of integrating by themselves a special branch of medical studies, the science of the constitution or constitutionology, requires us to dedicate special attention to it, presenting, even if only in its broad general guidelines and from the point of view of pathology and clinical practice, the main aspects and questions it raises.

Fig. 24.20 Tetanic facies.

When we comparatively observe the different individuals, we soon appreciate that, although they maintain the similarities that bring them together in a given species, they present great differences, in such a way that it can be assured that no two are exactly alike. This is proven, both in its external appearance and in its different systems, as well as in the anatomy and histology of its organs and tissues.

If there is such a visible and marked individualization in the morphological and anatomical, it is necessary to conclude that similar differences must exist in the organic functions and in the psyche, that very important and specialized function, developed to the highest degree in the human species and in which it finds its most complete expression, the individual personality.

If this is so in the anatomical, physiological and psychological, in the physical or somatic, as in the emotional or spiritual, it will be understood that both in pathology and in the clinic, attention must be paid to the special somatopsychic characteristics of the patient, to his individual biological constitution, which clearly distinguish and separate him from other individuals affected by the same morbid process, and which, without a doubt, must be a primary factor in his greater or lesser ease. to contract that or another disease, that is, in their predisposition for diseases, as well as in the degree of organic resistance that they offer.

From here we can easily extract the primary concept of what constitution is in medicine, considered by Brugsch, correctly, as the way a person's body is formed, which makes the concept of constitution coincide with the individual, since each individual has his or her own constitution. The study of the constitution, thus proposed, will, therefore, be that of the person considered as a whole and as an isolated individual, including in it, that of its physical and psychological constituent factors, that is, that of its bodily and mental qualities during its life, insofar as they are important for medicine.

This is how Brugsch and Kraus have proposed what is known in medicine as the theory of the person, which marks for many a new philosophical direction in this science, the neovitalist direction, which also entails a return to the consideration that since ancient times was given to the terrain in which the disease develops, already studied by Hippocrates, the Father of medicine and unduly abandoned in

later periods when the development of the mechanistic conception

It will not be difficult now to understand the extraordinary importance acquired by the studies of individual constitution in medicine, both from the point of view of diagnosis (constitution and predisposition) and prognosis (constitution and individual resistance against the disease). It will not surprise us, either, that in the second half of the 20th century there has been a strong trend in favor of constitutional studies, orienting itself towards an approach to the disease from the personal, individual point of view, removing it from the insecure terrain of abstraction that the concept of the disease means to place it in the firm and safe field of the study of the patient, the primary and only source of all pathological studies.

On the other hand, if the sagacious and truthful popular observation had always highlighted the importance of the individual constitution in pathology, and sharply distinguished the existence of a 'strong constitution' and a 'weak constitution' in 'healthy' subjects and in 'sick' subjects, respectively, consecrating the value of the somatic in constitution, the painful experience of the world wars clearly revealed the importance of the psychic component of the constitution, evidencing that - as Dr. Jimena de la Vega rightly says - somatic robustness was not enough to withstand the hardships and sacrifices of that front, since men with better organic appearance appeared to be victims of terror neuroses and with very complex pictures of functional disturbance, with somaic fixation; on the other hand, many others with true organic lesions were able to overcome, as it was not possible to suspect, the anguishing moments of that conflict.

It is, therefore, essential to establish a correct diagnosis and formulate an accurate prognosis to take into account the individual constitution of the patient, his psychophysical unit, since his morbid predisposition and his resistance to the etiological agent that attacks him will fundamentally depend on the inseparable system of these reactions.

The morphological, functional and psychic characteristics of the individual constitution are due to the action of two main groups of factors: internal factors and external constitutional factors.

The internal factors of the constitution are none other than hereditary ones, called genotypic, responsible for transmitting the specific characteristics of the species from generation to generation.

External factors, also called paratypic or non-hereditary, are found in the environment in which the individual develops, which powerfully influences its constitutional development, imprinting numerous characters on it.

The set of internal genotypic or hereditary factors responsible for hereditary constitutional characteristics is called genotype or idiotype, which can be defined by saying that it is the material substratum responsible for transmitting and preserving the invariable permanence of the species through space and time.

On the genotype of each individual, that is, on their hereditary constitutional characteristics, current or potential, the environment acts, in a clear and indisputable way, that is, peristasis, in which external or paratypic factors are found, determining more or less intense constitutional modifications according to the individual adaptive capacity. These adaptive modifications that external factors produce in the genotype and that can be seen in anatomical changes (shape, size, color, etc.) and psychic changes (way of behaving and reacting, character, etc.) are what make up the organic paratype, which can be defined, therefore, as that part of the constitution due to the reaction of the genotype with the environment.

The fact that genotypic or hereditary factors fulfill the important function of preserving the main characteristics of the species does not exclude the occurrence of variations in them, especially individual, and unrelated to environmental influences. To understand the possible existence of a wide hereditary constitutional variation, it is enough to remember the elementary knowledge of heredity, especially the distribution of the different genes on the chromosomes, the loss of hereditary characters that the reductional partition implies and the large number of possible different combinations between the chromosomes of the two gametes that conjugate.

From the reciprocal action and influence of both groups of constitutional factors or components - internal or genotypic and external or paratypic - arises the individual constitution, which is, more than a sum or a product of both factors, the result of a constant interaction of these.

In reality, what we appreciate as the constitution of an individual is his genotype with the paratypic modifications that external or environmental factors have imprinted on him. Therefore, the result of the reciprocal influence of genotypic and paratypic factors, in its objective, external and internal expression, is called a phenotype.

Schematically it is represented as follows:

GENOTYPE + PARATYPE = PHENOTYPE

From all that has been said, it will be easy to understand that there can be equal phenotypes with different genotypes and different phenotypes with the same genotypes. Thus, it is seen that the red-flowered variety of the Primula sinensis plant gives white flowers when placed at the temperature of

35 °C, different from its usual environment which is 10-15 °C, making it impossible to distinguish it from the pure white variety under these conditions. Here the influence of an external or paratypic factor is clearly seen modifying the phenotype of the plant, without altering its genotype, since, if it is brought back to its usual room temperature, it flowers again in red. In animals, in rabbits, for example, similar changes have been observed, changing the skin color from white to black, and vice versa, under the influence of temperature. In humans, differences determined by the two types of factors mentioned are also observed.

As a synthesis of the above and to establish, as best as possible, the concept of constitution that exists in the different schools, we are going to refer to some of the definitions that have been given of it.

Kraus considers that the constitution, as a problem of general pathology, is defined by the inherited and acquired characters of the individual, analyzed morphologically and functionally both with respect to the behavior of certain partial functions and to the sum of the properties derived from the activity of the body and spirit in relation, mainly, to the organic way of being and with respect to its demands for resistance, rejuvenation and vitality.

Brugsch defines it more clearly, considering that in medicine the constitution is the psychophysical relationship determined by the unity and totality of a vital system whose internal conditions in combination with the external ones (external world, social position, environment, etc.) are in constant balance.

Pende, the main figure of the Italian school, defines the constitution as the morphological, physiological and psychic result – variable from individual to individual – of the properties of all the cellular and humoral elements of the body.

The purpose of the study of the constitution in medicine is, as has been said, the search for individual characteristics that can serve as a basis for the diagnosis and, above all, for the prognosis of patients.

The study of the constitution has used two main methods. One, the oldest, used intuition, classifying the different people in a population into constitutional types that are expressive even for the layman. This is what is known as typology or phenotype. Another, more exact, method is based on body measurements: it is somatometry. Finally, an attempt is made to complete the anthropometric results with all those characters that, such as the person's posture, skeleton, hair or eye color, cannot be expressed quantitatively. This constitutes somatoscopy. The set of these procedures

Cough allows us to acquire a fairly complete concept of the person's morphology.

Typology or phenotype

In almost all countries and based on the most salient differences in body architecture, morphological or typological classifications have been established. They are the so-called Italian classification (De Giovanni, Viola, Pende); French classification (Sigaud, Chaillon, Mac Auliffe) and the German classification (Kretschmer).

Italian classification (De Giovanni, Viola and Pende)

The classification of the Italian school recognizes as a basis, ontogenically, two laws: one that determines the increase in mass or weight increase, and another that determines corporeal differentiation or evolution of form. The mass or weight finds its expression in the trunk (representing the vegetative system) and the shape in the extremities (representing the relationship system).

Viola, comparing the proportions of the trunk and the limbs, formulates the following morphogenetic law: weight increase or mass increase, and morphological evolution, or corporeal differentiation, are ontogenically in inverse proportion. That is, when there is an increase in mass (trunk) there is little morphological evolution (limbs), and vice versa; That is, individuals with a highly developed trunk generally have short limbs and are opposed to those with long limbs who almost always have a reduced trunk.

This constitutes the fundamental basis of its constitutional types. When both elements, mass and morphological evolution, are balanced, a well-proportioned adult organism results: the normotype or normolinear. When these factors (mass or trunk and shape or limbs) deviate from the normal measurement in one direction or another, due to excessive development of weight or mass (trunk) or due to too much morphological differentiation (limbs), the two large categories of normal human constitutional types arise: one, with exaggerated development of the trunk, is the brevilineal O megaloplanchnic; another, with exaggerated development of the limbs, is the longilinear or microsplanchnic. The first, Pende also calls hypervegetative, because in it, the trunk, with its vegetative functions, reaches the greatest development, while the second he calls hypovegetative because in it it is the members, representatives of the relationship system, that acquire greater importance.

In the former, anabolism and endocrine glands will predominate with anabolic hormones that act synergistically with the parasympathetic hormone; In the latter, catabolism and exciting catabolic hormones from the sympathetic nervous system will prevail.

We will now study, in summary, the most salient characteristics and the main predispositions of the constitutional types of the Italian school.

Normotype or normolinear

The morphology of these individuals reflects the balanced intervention of the internal and external factors already mentioned. They are well proportioned, with an average development of their viscera and a good endocrine-vegetative balance, which culminates in the good balance of their psyche and morals. They vary, as is understandable, in different races and countries, and generally have a size equal to the wingspan, that is, the distance between the tips of their middle fingers when their arms are extended in a cross. The height is also equal to twice its chest circumference. It is interesting to note that many eccentrics and criminals present this constitutional type.

Longilinear or microsplanchnic

In this morphological type (also called hypovegetative) a disproportionate development of the limbs and a small, reduced trunk are observed. This type is rather tall and thin, with a predominance of vertical dimensions. The skull is dolichocephalic, the neck is long and thin, like the trunk, in which reduced anteroposterior dimensions are observed. The bright, affectionate look and the large, slanted eyes. They pale and blush easily. The thorax appears flattened in an anteroposterior direction and concave forward, which would be well appreciated if it were examined by making a horizontal cut in its upper portion. The epigastric angle is acute, closed; the second protruding rib and the tenth floating.

Their muscles are generally poorly developed, with a certain hypotonia, which causes them to have winged scapulae, that is, separated from the thorax, simulating wings. The organs reach little development, generally presenting the minimum normal dimensions. There is a tendency to ptosis or descent of the viscera. When examined radioscopically, the small heart is observed, hanging from a narrow aorta, elongated like a heart in a drop. The diaphragm, hypotonic, is lowered and the amplitude of its movements decreased during breathing. This low position of the diaphragm favors ptosis of the abdominal viscera. The teeth of these subjects are generally well established, bluish-white in color and prone to cavities.

The general appearance of longiliners is rather ungainly and they are passionate, emotional and lovers of art in all its manifestations.

When all the facial characteristics of this constitutional type are found together in an individual, especially the female sex, it corresponds entirely to mind with that described by Landousy, with the name of "morbid beauty" which, although it is not always linked to a beautiful face, is beautiful because of its intelligent and affectionate look.

tuosa.

The morbid predispositions that this morphological type presents depend essentially on its thoracic anatomy. The narrow thorax, and, according to Bauer, the extraordinary length of the lung, which increases the distance from the hilum to the apex of the lung, predisposes it to pulmonary tuberculosis and its apical location. It is clear that this does not mean that all longline animals are tuberculous, but it is observed that they have a lower resistance to it. They also have a certain predisposition to nervous diseases and hyperthyroidism, which is explained by the predominance of the sympathetic and thyroid in their constitution.

On the contrary, diabetes, gout, chronic rheumatism, nephritis and diseases of the heart and vessels are very rarely observed in them.

In this type, it is interesting to highlight the great diagnostic value that any thickening and densification of the aortic radiological shadow acquires, which is narrow and of low density.

It should be mentioned that there are some special variants of interest of the longilinear type of constitution. One of them is what is known as the Venetian type, observed especially in women in whom, to the already described characteristics of this constitutional type, are added, making it unique, the extraordinary whiteness of the skin, frequently dotted with freckles, the saffron red hair - almost always curly - the greenish eyes and the very white and bluish teeth. In these individuals, tuberculosis is generally extremely serious, and it has not been possible to satisfactorily explain this constitutional inferiority.

Another variant of interest is represented by Stiller's asthenic constitution or asthenic habit, in which, to the features that characterize the longilinear phenotype, always in these very accentuated cases, is added, as an essential characteristic, for Stiller the tenth floating rib, a symptom around which the rest of the constitutional anomalies are grouped. They exhibit to a high degree the predispositions of their constitutional type, especially neurasthenic-type nervous disorders and digestive disorders, related more to the abdominal visceral ptosis they present than to their neuropathic defect (fig. 24.21).

Brevilinear or macrosplanchnic

In this constitutional type (also called megalosplanchnic or hypervegetative), which corresponds to what was formerly known as an apoplectic constitution, a well-developed trunk is observed.

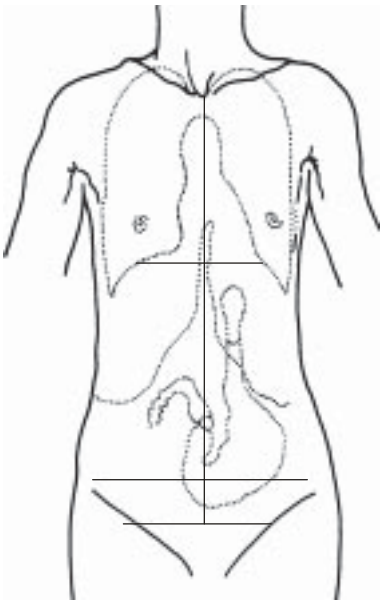


Fig. 24.21 Stiller's asthenic habit.

do, with short and thick limbs. These individuals are generally short and corpulent. The predominance of the development of their trunk in relation to that of their limbs makes them appear tall when they are sitting and short if they are standing. They are those thick individuals, with short necks, who seem to be " overflowing with health", who popular observation points out, not without reason, as prone to congestion and cerebral hemorrhages.

The skull is brachycephalic, the neck is wide and short, and it sometimes appears that the head rests directly on the trunk. The thorax is wide in all its diameters, with a very open, obtuse epigastric angle. The muscles are well developed and have a normal or increased tone.

The viscera are located in the normal position, they reach their maximum development, especially in the cardiovascular system, where, especially in the radioscopic examination, a large tonic heart with predominant transverse diameter is observed, resting on a high diaphragm, of good tone, which gives rise to large respiratory expansions. The aorta is thick, with great radiological density, so much so that, if these constitutional characteristics are forgotten, it frequently leads to the erroneous diagnosis of aortitis. Their teeth are strong, regular, with great resistance to cavities.

In short, it is a constitutional type of great strength and vital capacity, with magnificent respiratory and circulatory function, good appetite and better digestion, endowed with powerful reproductive capacity. They are energetic men, with great fighting capacity.

The morbid predispositions presented by individuals carrying this constitutional type find their reason for being fundamentally in this exuberance of vitality with which they have been naturally endowed.

Excesses of all kinds lead them to contract different conditions, such as venereal diseases and nutritional diseases (diabetes, obesity, gout, etc.). They frequently suffer from chronic rheumatism, chronic nephritis, high blood pressure, arteriosclerosis, cerebral hemorrhages and thromboses, lithiasis, etc.

They are resistant to tuberculosis, which, when it occurs in these types, generally adopts a slow, torpid evolution.

French classification (Sigaud, Chaillon and Mac Auliffe)

The French classification of Sigaud and his disciples Chaillon and Mac Auliffe, also accepted by the Viennese school, adjusts to clinical observation data, without using anthropometric measurements, and distinguishes four constitutional types that it calls: muscular, respiratory, cerebral and digestive (fig. 24.22).

Sigaud starts his classification from a principle of morphological development that serves as its basis. Think that from the moment of fertilization, a process of cell division takes place in the organism from the center to the periphery, which stops the moment the central force is consumed. From this moment on, the cellular conglomerate has only the energy necessary to maintain its life, producing differences with the center on its periphery, influenced by the outside world. This is represented by the cardiovascular-renal system, without contact with the outside world, while the periphery is represented by the respiratory, digestive, nervous, muscular and sexual systems, in intimate relationship with the environment. Hence, depending on whether one of these systems predominates in the development, the four types mentioned in which the different individual constitutions are classified here are distinguished.

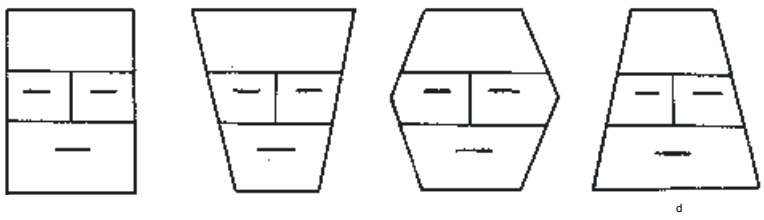


Fig. 24.22 Scheme of the four fundamental facial types (French classification): a, muscular facies; b, cerebral facies; c, respiratory facies; d, digestive facies.

We will study the main characteristics of the types of the French classification. In their study, the first basis for classification is the division of the head into three segments, limited by planes that pass through the root and base of the nose. Thus, an upper or cephalic segment is constituted, a middle or respiratory segment, and a lower or digestive segment, the predominance of which characterizes the cerebral, respiratory and digestive types, respectively, with the muscular type presenting equal magnitude in the three segments.

muscular type

It generally presents a brachycephalic skull, harmoniously constituted; The three segments have the shape of a square. The line of insertion of the hair on the forehead is usually straight and on both sides it also forms a right angle, which distinguishes it from the digestive type in which the hair is implanted forming an arc and from the cerebral type, in which it forms an incoming angle on the sides. The eyebrows are low and straight and the hair is abundant. The trunk is well formed, without prominences of the abdomen, with an epigastric angle of medium opening. The shoulders are wide and high and the muscles are strongly developed in the extremities. It corresponds to the type of classical beauty and athletes and criminals can be seen in it.

Respiratory type

It presents a special development of the thorax and the segments of the skull and face that serve for respiration. The thorax is very long, so that the last ribs reach almost to the iliac crest and the epigastric angle is acute. The segment of the face between the base and the root of the nose is more developed than the other two, the nose being generally large, long and often curved. The breadth of the maxillary and frontal sinuses and the marked development of the zygomatic processes give the face of these subjects a hexagonal appearance.

Brain type

It is characterized by a certain disproportion between the gracefulness of the body, which is fine and delicate, and the skull, which is notably large, especially with exaggerated development of the frontal region. The great development of the forehead means that the upper segment of the face, located above the plane that passes through the root of the nose, predominates over the other two, giving the face a pyramidal shape with its vertex directed downwards. This type is complemented by the characteristic insertion of the hair with its large lateral entrances, large ears, like the eyes, generally very bright, and small limbs and thorax. The cerebral ones are the representatives of intelligence.

digestive type

Here the lower segment of the face is highly developed, located below the plane that passes through the base of the nose, which clearly predominates over the other two segments. The distance between the base of the nose and the chin is very large, which, together with the exaggerated development of the branches of the lower jaw, gives the face a pyramidal appearance with the base in the lower jaw and the vertex in the occiput. The neck is short and the thorax is wide and short, with an always obtuse epigastric angle, a very low navel and a great separation between the iliac crest and the last ribs. The abdomen is very developed and prominent. Individuals carrying this type are generally obese.

The muscular type of the French classification almost always corresponds to the normotype or normolinear of the Italian classification. The French digestive type is almost always observed in short-line individuals, and the respiratory and cerebral types are more frequently observed in long-line individuals.

Studying the morbid predispositions of the different types of the French classification, Bauer has found that the respiratory type is much less susceptible to tuberculosis than the cerebral type. Syphilitic aortitis and kidney diseases are seen more in the digestive and muscular types. Among the cerebral ones, neurasthenia and hysterism frequently occur, and rheumatism attacks the muscular type.

German Kretschmer classification

Carrying out studies of the body architecture of mentally ill patients, Kretschmer establishes one of the most interesting typological classifications, with the merit of bringing into play the psychic aspect of the constitution, specifying the affinities between the different constitutional morphological types and the psychic types that he designates as cycloid and schizoid temperaments.

Kretschmer distinguishes the following constitutional types: athletic, asthenic-leptosomal, pycnic and dysplastic (fig. 24.23) .

athletic type

It is characterized by the great development of the skeleton and muscles. It would correspond to the muscular type of the French classification, and has an elongated ovoid face.

Asthenic-leptosomal type

(The first name is reserved for the most pronounced types.) It corresponds, in general terms, to the longilinear type of the Italian classification. It has an ovoid face with a short axis.

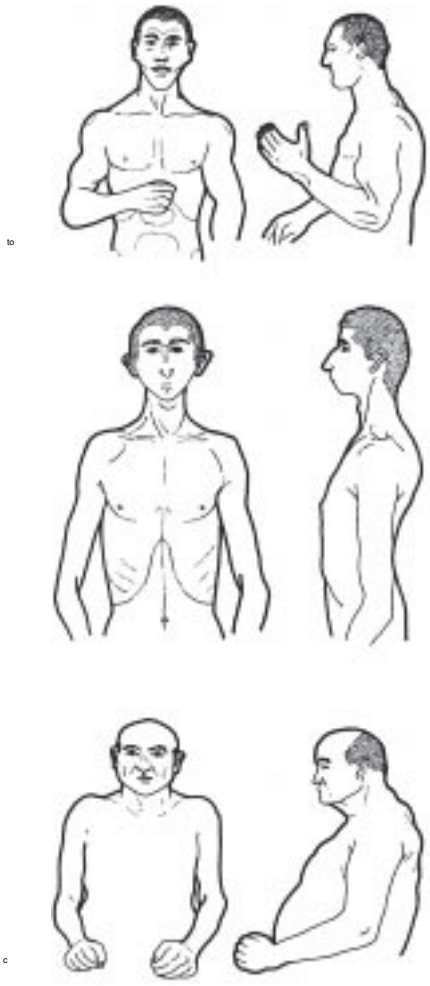


Fig. 24.23 Some examples of Kretschmer's classification:
a, athletic; b, asthenic-leptosomal; C, picnic.

picnic type

It is characterized by the great capacity of the visceral cavities (head, thorax, abdomen); Its general appearance coincides with the brevilinear type of the Italian classification. The face tends to have a shape called pentagonal. The appearance of well-marked cases of this type is, according to Kretschmer, very characteristic: short stature, with a wide face, short and solid neck; large, thick belly, and wide, deep, domed thorax. The neck in the picnic does not follow a straight line, but rather an oblique line and is confused with the upper end of the sternum.
These individuals have a tendency to accumulate fat, especially in the pubic area, on the trunk and diffusely on the face, hips and knees. It would correspond to the digestive type of the French classification.

dysplastic type

It differs greatly from the average and presents glandular disorders. This type includes, for example, eunuchoid gigantism, masculinisms, obesity eunucoid and polyglandular and the infantile and hypoplastic types tics.

Psychic aspect of Kretschmer's classification:
cyclothymic and schizothymic temperament

As we already said, the extraordinary merit of this constitutional classification consists, mainly, in having introduced the psychic factor. Kretschmer affirms that between his asthenic-leptosomal, athletic and dysplastic types and the psychic type that he designates as schizoid temperament, there is great affinity, which also occurs between the psychic type called by him the cycloid or circular temperament and the pyknic constitutional type.

Kretschmer came to individualize these psychic types based on meticulous anthropometric studies carried out on mentally ill patients classified, according to Krapelin's psychiatric criteria, into the two large groups of schizophrenics and circular or manic-depressives. From these studies he deduced that the body and temperament (temperament is the dynamic aspect or reactive form of the constitution, as opposed to habit, that is, the morphological or static expression of the same) are in close biological correlation established through the intermediary of the endocrine glands, that between circular or manic-depressive madness and the pyknic habit there is a marked affinity and that, also, there is a close relationship between schizophrenia and the asthenic, athletic and dysplastic.

As can be seen, in this case, Kretschmer used a method frequently applied in medicine: starting from the pathological to know the normal. In fact, both schizophrenia and manic-depressive psychosis are psychologically the pathological representatives of the two large temperamental groups into which we can classify individuals that we consider mentally normal. These groups are: the schizothymic temperament and the cyclothymic temperament respectively. Between them and the frankly pathological states of schizophrenia and circular mania are schizoids and cycloids, types that temperamentally oscillate between health and illness, without being able to be truly considered healthy, but not being truly sick. The schizoid oscillates between the schizothymic temperament and schizophrenia, and the cycloid is between the cyclothymic and the circular or manic-depressive. Its graduation could be schematized in the following way:

schizothymic	Cyclothymic
Schizoid	Cycloid
Schizophrenic	Manic-depressive circular

People with a cyclothymic temperament are happy, kind, welcoming and sociable, easy to make friends with. Very cordial, they respond easily, bluntly and appropriately, to the stimuli of the environment, finding every feeling a great resonance in their spirit. They oscillate between exaltation (joy) and depression (sadness), sometimes one of these two spiritual states predominates, which gives them a happy or sad temperamental tone, but always discovering in them some depressive component if they are happy, or some spark of good humor if they appear sad; It is this combination of both components that Kretschmer calls the diathetic proportion. Their ease of relating to the environment clearly corresponds to what Bleuler has called "'tuning'."

Schizothymics are the antithesis of the cyclothymic temperament. To their simple and transparent way of being, they oppose a complicated temperament that is difficult to understand and capture. There is always a surface and a background in them that they jealously care to display. They do not let anyone get inside them, living for themselves and within themselves, a characteristic that Bleuler has named autism. They almost never become known, not even to their family, due to the hermetic barrier in which they enclose themselves. They oscillate between hypersensitivity (irritability) and anesthesia (coldness). They frequently react inappropriately to environmental stimuli. On the surface they seem brutal, sullen, ironic and biting, and on the intimate level they appear, deeply, either cold and disaffected or delicate and sweet. Opposite feelings tend to occur in their spirit at the same time and this is what a schizophrenic German playwright said: "I am cold as ice, and yet I am overwhelmed by an overflowing tenderness."

The mixture of these two temperamental factors is called psychoesthetic proportion by Kretschmer. Schizotymics are generally unsociable, with a tendency to isolate themselves in any activity. It is interesting to highlight the complexity that this temperament usually presents in everything related to sexual matters, which contrasts with the simplicity and naturalness with which it develops in cyclothymics.

As can be understood, in the normal individual both temperamental elements act more or less proportionally. The predominance of one of these variants gives rise to the corresponding temperaments: schizothymic or cyclothymic. The exaltation of one of them corresponds to the schizoid or the cycloid and, finally, when it becomes morbidly acute, and the balance with reality is broken, one falls into the pathological states of schizophrenia, or circular or manic-depressive madness. Kretschmer rightly says that Psychopathies are nothing more than the caricature expression of normality.

Somatometry

As we already said, it is another of the main methods used in the study of the constitution. It is more exact and there is no doubt that better than any typology, it is the classification of individuals according to body measurements, the study of which precisely integrates somatometry.

Somatometry, which should never exclude the typological classification of the subject, uses different body measurements, indices and coefficients that will be more specially studied in endocrine semiology. Here we will limit ourselves to stating the most important ones.

Among the somatometric measurements we study height, chest circumference or thoracic perimeter, trunk length, shoulder width, pelvis width, and the length of the arms and legs. Among the cephalic measurements, the horizontal perimeter of the head, its maximum length and width, the width of the cheekbones and the physiognomic and morphological height of the face are of particular importance.

The chest circumference is measured at the level of the nipples, with the arms in abduction; the length of the trunk, from the vertex of the skull to the line that passes between the ischial tuberosities; Shoulder width is the distance in a straight line between one acromion and the other and the width of the pelvis is the distance, also in a straight line, between both anterior superior iliac spines.

Indices and coefficients are used to compare different people with each other. We will mention only two: the Pignet index and the Rohrer index.

The Pignet index is determined by subtracting from the figure corresponding to height in centimeters, the sum of the values of the chest circumference in centimeters and the weight in kilograms.

Index by Pignet: Height - (Chest perimeter + Weight) (cm) (cm) (kg) Individuals of medium build, normal, have a Pignet index between 21 and 25. This index is below 10 in very strong individuals and above 35 in weaker ones.

The Rohrer index gives a measure of corpulence and is calculated, according to Horster, by dividing the product of weight in kilograms multiplied by 100 by the product of height in centimeters multiplied by three.

$$\text{Rohrer index} = \frac{\text{Weight (kg) } 100}{\text{Height (cm) } 3}$$

The index increases in very strong individuals and decreases in very weak ones.

Somatoscopy

With this method, an attempt is made to complete the typological and somatometric data, with all those characters that cannot be expressed quantitatively. In this way, the posture of the body in the standing position, the musculature, the adipose tissue, the skeletal system, the complexion and shape of the hair and hair of the subject are studied. In addition, attention is paid to racial signs and the shape of the chest.

Lloyd T. Brown's body posture and postural types were already described in Section I.

The somatoscopic study is always completed with the other data that we mentioned above. Thus, the examination of the muscles will be noted, classifying them, as the case may be, as very vigorous, good, medium, weak and very weak; The adipose panniculus will be observed, which will allow us to distinguish very obese, obese, normal, skinny and very thin individuals, the bone system, which will be very coarse, coarse, medium, delicate and very delicate.

The complexion is also studied, made up of a complex of inherited signs, represented by the pigmentation of the hair, skin and iris. Thus, we study hair color, for whose classification special tables are used in which the different colors correspond to numbers; the color of the skin, which is studied both in the places exposed to the sun (front) and on the trunk, and is also classified by special tables, and finally the color of the eyes that can be compared with the glass rods of Martín's ocular table, which varies from number 1 to 16, the brown colors of the iris corresponding to the first numbers (1-6), the blue ones to the last ones (13-16) and the intermediate ones to the central ones. (7-12).

The shape of hair (straight, wavy, curly, etc.) and body hair are also studied here, as well as the interesting racial signs that allow different types of races to be distinguished. The shape of the thorax, which will be studied by us in respiratory semiology, is also included as somatoscopic data.

Having made this review of the different methods that we have for the study of the individual constitution, and of their most important results, it will be understood that through them we can classify the individual well from the point of view of his constitution, duly valuing the valuable data that it provides for the diagnosis and prognosis of " the person", who is sick. Its inclusion in the clinical records will allow us, when we reread them over time, to animate them with the personality of the subject to which they refer, removing that cold impression, of lack of life, that they provide when they lack these constitutional data.

Neurovegetative aspects of the constitution (works of Ppingery Hess)

The importance of the role that the vegetative nervous system plays in the constitution cannot be ignored. It was not, however, until 1910, when Hans Eppinger and Leo Hess, from Vienna, published a monograph entitled Vagotonia, in which the constitutional neurovegetative component was given the importance it deserved. In contrast to this primitive ignorance, in later times it was given, on the contrary, exaggerated value, leading to the elaboration of schemes and divisions, more imaginative than real, which could not withstand the clinical test.

Studying nervous patients, especially those classified within the somewhat indefinite group of "neuroses", and applying to the clinic the conception of vagal tone established by experimental physiology, Eppinger and Hess described vagotonia, in which they grouped, in their own words, all the constitutional states in which, in addition to the manifestations linked to an increase in vagus tone and excessive irritability, an abnormal sensitivity to pilocarpine.

Eppinger and Hess described the subjects with the vagotonic constitution in the following way: they are, they said, young, middle-aged individuals, with banal cardiac, gastric or intestinal disorders, who resemble neurasthenics, and of whom an idea can be formed by saying that they are "invalids of the nervous system. They present frequent changes in the color of the face, turning pale and blushing easily, which which frequently constitutes a concern for the patient. When undressing, the same color changes appear on the chest and arms. The hands are purplish, cyanotic, especially the fingertips, and when palpated, they are moist and cold for the most insignificant reasons, either all over the body, or in certain regions, such as the back, head, face or feet, and it is very characteristic. During the clinical examination, sweat drops are observed falling from the armpit along the lateral plane of the thorax. The eyes are large, wide open and bright.

They swallow frequently, which means that their mouth fills with saliva, and they present a typical decrease in the sensitivity of the pharyngeal wall, such that a tongue depressor can be maneuvered on the soft palate and pharynx without provoking the usual reflexes.

Breathing is slow, deep and irregular, with a feeling of respiratory distress on many occasions, giving rise to a very characteristic type of breathing, reminiscent of a sigh, and which has been called respiration.

sighing sensation, often confused with organic dyspnea.

If we examine the circulatory system we find visible heartbeats in the neck and epigastrium; The irregular pulse has a variable frequency although, generally, there is bradycardia. Blood pressure is low, close to the minimum normal levels. Respiratory sinus arrhythmia is frequently found in them. They usually complain of precordial discomfort and a feeling of constriction of the heart, symptoms that often become serious worries that accentuate the patient's nervousness.

On the part of the digestive tract, the existence of a special sensation of food being stopped in the esophagus, or an esophageal ball, is observed; belching and feeling of fullness and distension after meals. They are aerophages. The constipation is almost constant and his stools are sometimes made up of scybales, and other times in the form of a ribbon, depending on the type of colic spasm existing. On other occasions they present diarrhea that cannot be explained by any dietary transgression.

Frequency is also observed in them, and their urine is rich in phosphates and oxalates. Vagotonics are sexually libidinous, with sudden and frequent but short erections, and rapid, premature ejaculation.

The tendon reflexes are increased and there is a slight tremor of the eyelids, tongue and extended fingers.

As can be seen, in this complete description by the Viennese authors, vagal or parasympathetic elements and sympathetic elements are discovered. Rather, it is a state of hyperexcitability and global vegetative hypertonia, which corresponds quite well to what was formerly described as nervous diathesis.

Eppinger and Hess would have already glimpsed a sympathicotonic state whose main signs they pointed out. Subsequently, as is known, the concept of the antagonistic balance of the two fundamental exponents of the vegetative nervous system was developed: the great sympathetic and the parasympathetic, attempting to schematize in the clinic the symptoms that manifested the existence of sympathetic or parasympathetic hypertonia.

In parasympathetic or vagal hypertonia (hyperparasympathicotonia or vagotonia), hyperhidrosis or abundant sweating, miosis, sialorrhea, thoracic or esophageal ball, spasmodic constipation, bradycardia, arterial hypotension, respiratory arrhythmia, and a marked increase in the oculocardiac reflex are preferentially observed.

In sympathetic hypertonia (hypersympathicotonia) the following cardinal symptoms are observed: tendency to exophthalmia and mydriasis, dryness of the skin and mouth, easy appearance of the phenomenon called meat of galina, dermographism, tachycardia, tendency to high blood pressure, intolerance to cold and, importantly, abolition or inversion of the Dagnini-Aschner oculocardiac reflex.

The observation of patients with vegetative disorders shows, however, that a classification such as this one, which only includes two antagonistic clinical conditions, vagotonia and sympathicotonia, leaves much to be desired, since it is impossible to classify a large number of patients who, at the same time, or successively, present sympathetic symptoms. This and the increasingly complete knowledge of the extraordinary complexity of the vegetative nervous system have been diminishing the exaggerated importance that was given to these schemes, causing a tendency to abandon the primitive concept of Eppinger and Hess and to try to replace it with others better adapted to our current knowledge. However, this does not diminish the importance of the Viennese classification, whose clinical and constitutional bases will always be of useful practical application.

Constitutional endocrine aspects

The importance of endocrine factors in the constitution is at least as great as that of neurovegetative factors. To understand it, it is enough to remember the decisive influence that the internal secretion glands exert on the physical and mental development of the subject. It is not surprising that considering the predominance that one of them may present in each case, and in an effort to make an analysis of the individual internal secretion formula, as Bauer wants, different endocrine constitutions have been studied.

This describes a thyrotoxic or hyperthyroid constitution, which includes individuals who are typologically long-shaped, hypersympathicotonic, with a high metabolism and generally enlarged thyroid. Everything seems to indicate the existence in them of thyroid hyperfunction, which in certain circumstances, inconsequential for other constitutional types, would explode into a picture of thyrotoxicosis, frankly pathological.

The hypothyroid constitution would generally occur in individuals with characteristics opposite to the previous ones, thick, small, with the characters already described in the brief lines, with a tendency to obesity, apathetic and sleepy, very sensitive to cold, with very little development of the external third of the eyebrows, dry skin, etc., and, finally, a decreased basal metabolism. The resistance of these subjects to infectious states and trauma is decreased, which must be taken into account when formulating the prognosis.

The acromegalic or hyperpituitary constitution is found in tall individuals, with a powerful skeleton,

with large and strong bones with a very developed and prominent lower jaw, as well as the browbone and cranial sinuses, a large and wide nose, thick lips and club-shaped limbs. This special constitution is explained by a hyperfunction of the eosinophilic cells of the anterior lobe of the pituitary gland and its exaggeration frankly falls into the realm of endocrine pathology.

The hypergenital constitution is characterized not only by a marked development of sexual characteristics, but also by the appearance of other very important symptoms. These are subjects who are ahead of their age peers in organic development and in their sexual characteristics, as well as in their psyche. They are those "very little men" or "very little women" children, as Viten says, who later turn out, generally and by virtue of the premature closure of their epiphyses, to be individuals with short legs, with strong muscles and a lot of hair on the trunk that contrasts with their premature baldness. Hirsutism is very marked when this is due to adrenal hyperfunction.

The hypogenital constitution is characterized by a deficient development of the genital organs and secondary sexual characteristics, and a thick type can be observed, with a large adipose panniculus, especially in the lower part of the belly and in the pubis, hips, chest and trochanters (eunuchoid obesity) and a tall type, with a marked predominance of its lower extremities, which are disproportionately long, as are the upper ones (gigantism). eunuchoid).

The hypoparathyroid constitution, due to a functional deficit of the parathyroids, mainly presents exaggerated excitability of the relational and vegetative nervous systems. This is observed when your nerves react to mechanical or electrical stimuli. They are those individuals who as children have suffered from the so-called spasmophilic diathesis with a tendency to suffer convulsive, tetanic or tetanoid attacks, laryngeal spasms and eclamptic paroxysms. They present alterations in the tooth enamel and bones, and blood calcium is greatly reduced.

They are predisposed to suffer tetany from any cause.

Finally, the hypochromaphin constitution, caused by a constitutional insufficiency of the adrenals, is characterized by arterial hypotension, weak pulse, bradycardia, easy fatigue and asthenia, hypothermia and muscular hypotonia. As Bauer correctly observes, general anesthesia (especially chloroformic), childbirth, surgical operations and acute or chronic infections are especially dangerous in these individuals.

Parcel inequalities of the constitution

Bauer raises the possibility of the existence of partial constitutional inferiorities, that is, limited to this or that part of the body, influencing, without However, in the general predisposition of the body for diseases.

This is how he distinguishes limited partial constitutional inferiority:

1. To one half of the body.
2. To the organs or tissues that come from one of the blastodermal leaves.
3. To certain systems, organs or parts of organs.
4. To an organic function or to all the cellular activities that intervene in said function.

As an example of the first case, a soldier is cited in whom the left half of the face was smaller than the right, the left testicle was smaller than the right, and the retina and pigmentation of the left eye were less developed than in the right. As an example of the second group, neurofibromatosis or Recklinghausen's disease is cited, originating from a constitutional disorder in the development of primitive ectodermal neuroepithelial cells. In the third group we find numerous examples, among which the constitutional inferiority of the stomach stands out, which predisposes it to gastric ulcer. As an example of the fourth group, renal diabetes can be cited, considered by many to be a functional degenerative stigma and characterized by a decrease in the renal excretion threshold for glucose, responsible for its appearance in the urine, with normal glycemia, a fundamental finding in this disease.

Constitution and provision

We have already said repeatedly that one of the primary purposes of the study of the individual constitution is to discover its possible morbid predispositions, that is, its disposition towards illness. Therefore, we must say two words about the problem of constitution and disposition (predisposition).

As is understood, predisposition entails the existence of an organic inferiority, of a constitutional anomaly, responsible for the decrease in individual resistance that explains the greater ease of contracting the disease.

Bauer groups these constitutional anomalies, differentiating them into evolutionary, morphological and functional.
The last two have been mentioned when referring to the territorial inferiorities of the constitution. The first are based on alterations in the evolutionary development of man, distinguishing cases that exhibit manifestations of infantilism and senilism (evolutionary constitution and involutionary constitution).

We consider it of greater interest to study here the main abnormal constitutions described, many of which have already been mentioned.

Thymolymphatic status, by Paltauf

It is an abnormal constitution characterized by enlargement of the thymus and hyperplasia of the entire lymphoid system (tonsils, lingual, pharyngeal and intestinal follicles, lymph nodes and spleen). Others of great interest are also found accompanying these fundamental anomalies of the thymolymphatic status, among which we can point out the poor development of the aorta and blood vessels, genital and chromaffin hypoplasia. Carriers of these constitutional anomalies present a very peculiar way of reacting, different from that of normal individuals.

Hypoplastic status, by Bartel

Here we also find hyperplasia of the thymus and lymphatic tissue and insufficient or defective development of organs and tissues that have a tendency to be invaded by connective tissue, a peculiarity that some French authors have described under the name of fibrous diathesis. Bartel does not consider the existence of hyperplasia of the thymus and lymphatic tissue to be essential for the diagnosis of hypoplastic status. Their predispositions are modeled on those of the thymolymphatic status.

Asthenic Constitution, by Stiller

It is another abnormal constitution whose characteristics and predispositions we learned about when studying the longilinear type of the Italian school.

Exudative diathesis, Czerny

In it, a great tendency to inflammation is observed, even due to the most banal stimuli. Carriers frequently suffer from colds, bronchial asthma, eye conditions, impetigo, eczema and mucomembranous colitis. They also present hyperplasia of the lymphoid tissue of Waldeyer's ring, with hypertrophy of the tonsils, adenoids, etc.

Neuropathic constitution

In it we find a special excitability of the relational and vegetative nervous systems. We recognize it by the exaltation of tendon and skin reflexes. neos, lability of the heart rate and hypertonia of the vagus and sympathetic. It is observed a lot among intellectuals and artists. This constitutional anomaly coincides in many points with the vagotonia of Eppinger and Hess.

arthritic diathesis

(Arthritis or herpetism.) It is an abnormal, hereditary constitution, observed mainly in subjects of the brevilineal type and characterized by the appearance in the individual and his family of certain diseases related to metabolic disturbances.

Obesity, diabetes, gout, gallstones and kidney stones, asthma, migraine, arteriosclerosis, eczema and other dermatoses are observed in them.

Serological constitution

There has also been talk of the existence of a serological constitution, attempting to establish a relationship between it and the predisposition to certain diseases. It is evident that the immunity that certain individuals present against some infecting agents may be due to a certain individual serological constitution. The study of the so-called blood groups also argues in the same sense, demonstrating that there are individual serological differences.

This very important topic of the constitution has been the subject of interesting experimental studies.

Constitution and temperament

Finally, everything seems to show that temperament, to which we alluded when studying the psychic aspect of Kretschmer's classification, is nothing more than the expression of a certain constitutional state and is as different in one individual or another as external habit can be. It is invariable like the constitutional state and accompanies the subject throughout life. Not even the best oriented educational efforts sometimes manage to modify the basic background of temperament; At most, they influence character. Popular wisdom already said it a long time ago in one of the most truthful Spanish sayings, "genius and figure, until the grave."

25

Introduction

The general inspection, in addition to the important data that we have already referred to in the previous chapter (ambulation, recumbency, attitude, gait, external habit and constitution, weight, height and appearance of the patient), also includes the examination of the skin and subcutaneous cellular tissue, which we will consider separately.

When studying the semiological data obtained by inspecting the skin we will limit ourselves to those that provide changes in its coloration and the study of its primary and secondary lesions, and for the specialized subject, Dermatology or Skin Diseases, we will leave the more in-depth study of these signs and others, also of great diagnostic value. We will also consider here, as a complement to the data provided by the inspection of the external integument, the examination of the phanera (hair and nails), the humidity of the skin and the so-called collateral circulation.

Semiotechnics

The skin and pharynx examination techniques were already pointed out in Chapter 3 of Section I. It is advisable that they be reviewed before undertaking the study of their alterations. However, we will remind you of the most important aspects.

Remember that the integuments must be examined in their entirety, discovering and observing, part by part, the body surface, always trying to have the necessary care and tact so as not to hurt the modesty and delicacy of the patient, because modesty and clumsy maneuvers alter the physical and functional conditions of the skin and psychologically predispose the patient against the doctor.

The integumentary inspection is complemented by the examination of the oral mucosa and ocular conjunctivae.

The following is explored:

1. General color and its regional variations: particularly in facies, extremities, genitals, parts exposed to friction or the elements (waist, forearms, etc.). For its interpretation, racial, temperamental, sex, age, seasonal, professional, etc. variations will be taken into account.

2. Surface: in its appearance, glossiness, humidity, peeling, folds, striae, trophic state, and elementary skin lesions, such as scars, nevi, ephelides, spots, etc.

3. Phanera, made up of nails and hair (hair, beard, lanugo, eyelashes and eyebrows): its characteristics and normal and pathological variants.
4. Venous circulation, venules and veins: their distribution, quantity, circulatory modality, type of networks they form, etc.

As a complement to the inspection, the doctor, to corroborate some data or to study others that are foreign to him, also uses palpation and diascopy.

Palpation should be superficial and light, or taking small folds between the thumb and index finger or other fingers of the hand. In this way it is studied:

1. Humidity and greasiness which, when not very evident, can escape simple inspection.
2. The surface, to appreciate it when it appears rough or dry, with roughness, peeling, or atrophic states.
3. Turgor and elasticity, the only way to appreciate them, since they depend on the thickness and resistance, which are only checked when making a fold.
4. Temperature, palpating symmetrical or neighboring areas or regions, and appreciating and comparing their respective temperatures.

Diascopy consists of observing a small area of the skin immediately after having been compressed with the index or other fingers of the hand; or even better, observe it through a spatula, a slide or a tongue depressor, made of colorless and transparent glass, with which light pressure can be applied.

With these methods, phlogistic, static or hyperemic congestive spots disappear while hemorrhagic or impregnation spots persist or become more apparent on the ischemic background produced by the pressure.

ELEMENTAL SKIN INJURIES

It is advisable to begin the study of skin alterations by analyzing the different types of primary and secondary skin lesions, which allows adequate recording of these and facilitates the understanding of the rest of the alterations.

PRIMARY SKIN LESIONS

1. Spot (spot) and patch. Non-palpable skin color changes, flat lesions (the color may be white, brown, black, red or purple). They differ only in size and their edges (fig. 25.1).

   Macula: <1 cm, circumscribed edges. Example: freckles or ephelides.

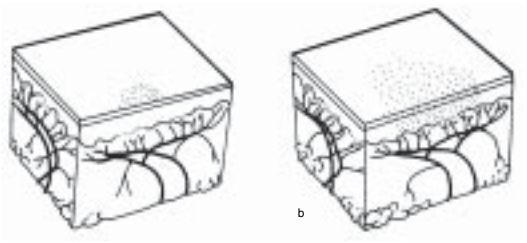


Fig. 25.1 Macula (a). Patch (b).

   Patch: > I cm, edges may be irregular.
   Example: vitiligo.

2. Papule and plaque. Solid, raised, palpable lesions, with circumscribed edges (Fig. 25.2).
   Papule: < 0.5 cm. Example: first acne lesions.

   Plate: > 0.5 cm. Example: xanthelasma of the eyelids.

   The plaques may be coalescent papules with a flat surface.

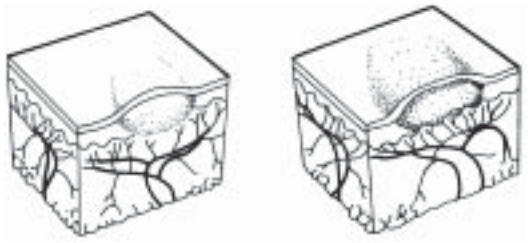


Fig. 25.2 Papule (a). Plate (b).

3. Nodule and tumor. Solid, raised, palpable lesions that extend deeper into the dermis than the papule and plaque. The nodules are circumscribed; Tumors do not always have a defined border (Fig. 25.3).

   Nodule: 0.5-2 cm. Example: sebaceous cyst.
   Tumor: > 2 cm. Example: neurofibroma.

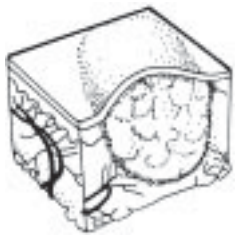


Fig. 25.3 Tumor.

4. Hive or wheal. Elevated lesion with transitory edges. Often irregularly. Varies in size and color. It is caused by the movement of serous fluid within the dermis. It is nothing more than localized, superficial edema of an area of the skin, somewhat temporary.
   Examples: hives and insect bites (fig. 25.4).

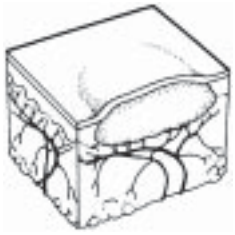
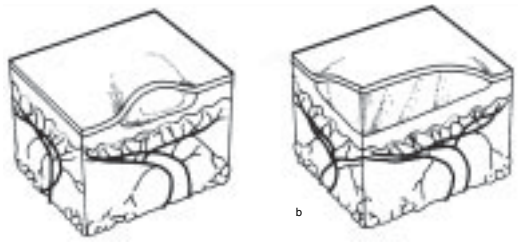


Fig. 25.4 Hive or wheal.

5. Blisters: vesicle, flictena and bulla or ampulla. Blisters are all raised, palpable and circumscribed lesions that contain serous fluid in a cavity located between the skin layers. Although the vesicle, flictena and bulla are all blisters, it is the bulla that many usually reserve the term bulla for (fig. 25.5).

Vesicle and flictena: <0.5 cm.

Bulla or blister: >0.5 cm.



b

Fig. 25.5 Vesicle (a). Bulla or blister (b).

The vesicle and the flictena differ in that the cavity of the former is multiloculated, which is why, when you puncture it, it is not completely emptied, only the liquid contained in the punctured locule comes out; while the cavity of the flictena is generally uniloculated, and like the bulla, when it breaks or punctures any part of its surface, it empties completely.

mind.

6. Pustule. It is a vesicle or blister filled with pus (Fig. 25. 6).

Fig. 25.6 Pustule.

7. Cyst. Semi-solid or fluid-filled lesion, but encapsulated, characteristics that differentiate it from the nodule and tumor, from the vesicle, the flictena and the bulla or ampulla. It is located in the dermis or subcutaneous cellular tissue (fig. 25. 7).

Fig. 25.7 uiste

SECONDARY SKIN LESIONS

1. Erosion. Loss of the superficial epidermis. It does not extend to the dermis. Humid, depressible area, which does not bleeds (fig. 25.8).

Fig. 25.8 Erosion.

2. Ulcer. Loss of deeper skin that extends past the epidermis. Loss of necrotic tissue that may bleed and leave scars (Fig. 25.9).

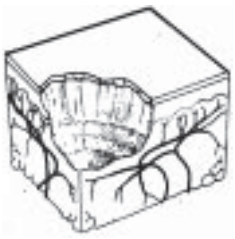


Fig. 25.9 ulcer.

3. Fissure. Linear crack or split in the skin. It can extend to the dermis (fig. 25.10).

Fig. 25.10 Fissure.

4. Scales. Secondary flakes of dead, whitish, decaying tissue. The flakes can stick to the skin. Color varies (white or silver) . The texture varies (fine, coarse) (fig. 25.11).

Fig. 25.11 Scales.

5. Scabs. Dried residue of serum, blood or pus, on the surface of the skin (fig. 25.12). A very adherent scab is a scab.

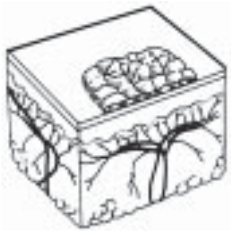


Fig. 25.12 Scab.

6. Scar. Mark left after healing a wound or injury. It represents the replacement of injured tissue with connective tissue (fig. 25.13). Young scars: purple or red. Mature or old scars:
bright white or pearlescent.

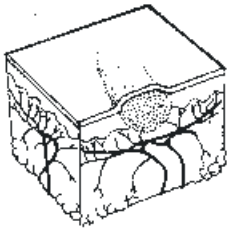


Fig. 25.13 Scar.

7. Keloid. Hypertrophied scar tissue, secondary to excessive formation of collagen tissue. Elevated, irregular, red (fig. 25.14). High incidence in black people.

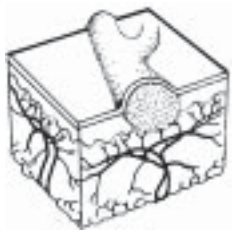


Fig. 25.14 uelooid.

8. Atrophy. Transparent, dry and thin appearance of the epidermis. Loss of surface markings secondary to loss of collagen or elastin. The underlying blood vessels can be seen (Fig. 25.15).

Fig. 25.15 Atrophy.

9. Lichenification. Thickening and wrinkling of the skin. Marked skin marks (fig. 25.16). It can be secondary to repeated irritations, traumas, contacts.

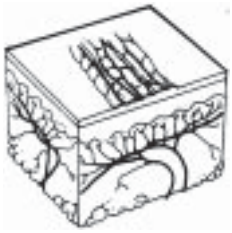


Fig. 25.16 Lichenification.

CHANGES IN SKIN COLOR

Skin color depends on two main factors in men:

1. From the red color of the blood, which circulates through the capillary networks of the dermis and is transparent through the thin layer of the epidermis.
2. From the black coloring matter or melanin found in the cells of the deep layer of the epidermis.

The red color of blood is due to the hemoglobin contained in red blood cells or red blood cells. The skin pigment, melanin, accumulates in the form of granulations in the basilar layer and in the stratum malpighiano of the epidermis, its presence being observed in both white and black races, only in the latter the pigment granulations are both much more voluminous and numerous.

As can be seen, the color of the skin depends on the quantity and quality of the blood that circulates in the capillary networks of the dermis, the number and caliber of the blood vessels in these networks, the melanin pigment of the epidermis and the greater or lesser transparency of the superficial or epidermal layers of the skin.

It will be easy to understand now that if in pathological circumstances the quantity or quality of the blood, the number or caliber of the blood vessels of the dermis, the quantity or distribution of melanin are modified, changes will appear in the coloration of the skin that will also

They can be observed in those cases in which various pigments are deposited in it, or vary, making the superficial layers of the epidermis more or less thick, through which the main chromatic factors responsible for the coloring of the skin are transparent.

Likewise, it can be seen that it is in the white race where the study of abnormal skin colors will be easier, which will be very difficult, or impossible, in other races, especially the black race.

Among the pathological discolorations of the skin, we will study, as the most important, the following: pallor, ruddyness, cyanosis, yellow discoloration (jaundice, especially) and the main pigmentary alterations: melanoderma, acromias, vitiligo, etc.

PALE

Concept

In paleness the rosy tint of the skin is absent;

This is white in color, sometimes with a slight yellowish tinge. The mucous membranes are also observed to be discolored, more or less bloodless; what can be seen in the lips, mouth and conjunctiva.

Semiognesis or pathophysiology

Paleness appears whenever the pink component of the skin color is reduced or absent. Since this depends on the red color of the blood circulating in the dermal capillaries, transparent through the epidermis, there will be paleness whenever the quantity or quality of blood, the caliber or number of the blood vessels in the dermis is altered or the epidermis increases in thickness or decreases its transparency, interfering with the passage through the red color of the blood, which in this case may be normal.

semiography

1. Paleness can be generalized throughout the body or localized to a part of it or to a limb, for example, a portion of it.
2. According to the intensity of the coloring, a light, medium or intense paleness is described.
3. According to the special tint they present, alabaster pallor and waxy pallor are distinguished, which seem to correspond to the special coloring of alabaster and wax, observed, above all, in certain anemic states; straw pallor, which presents a special yellow color, similar to straw, observed especially in cancer patients (straw tint of cancer patients); the greenish pallor, observed especially in chlorosis (a special type of anemia, which is where it gets its name, from chloros: green) in which the pallor has a very characteristic greenish tint and, finally, the pallor earthy or earthy, which is characterized by dark pigmentation, especially on the face, and which is observed in malaria and some liver diseases.

When the paleness has a yellowish tinge, it is necessary to distinguish it from forms of jaundice, that is, from what we know as sub-jaundice. To do this we will pay attention to the color of the sclera, which will appear more or less frankly yellowish in light jaundice, while it will preserve, or accentuate, its normal whiteness in pallor. The investigation of the other signs that make up the jaundice syndrome and the study and demonstration of the cause of the paleness will complete the differential diagnosis.

Semidiagnosis

Let us point out, first of all, the existence of a type of congenital or constitutional pallor, in which the pale color of the skin depends on a lower transparency, or a greater thickness of the superficial epidermal layers, or a decrease in the number of blood vessels that make up the dermal plexuses. This congenital pallor is not actually pathological, and is opposed to other types of acquired pallor, dependent on a morbid disorder. We should think about congenital pallor when we observe a pale individual with well-colored mucous membranes.

Paleness due to blood disorders

In other cases, paleness is due to pathological changes in the quality or quantity of blood circulating through the skin vessels. This can be observed when the amount of hemoglobin is decreased (oligochromemia), or when the number of red blood cells is below the normal number (oligocythemia), or when the blood mass has decreased due to external or internal bleeding (oligohemia).

Pallor due to oligochromemia is observed in all forms of anemia that lead to a decrease in the hemoglobin content of the blood with a more or less accentuated decrease in the number of red blood cells per cubic millimeter.

Pallor due to oligocythemia occurs in those forms of anemia that, without prejudice to the sometimes marked decrease in the amount of hemoglobin, are characterized by a marked decrease in the number of red blood cells per cubic millimeter.

Pallor due to oligohemia is found after large hemorrhages, external or internal, and is due to a decrease in blood mass. It is of great value in the diagnosis of internal bleeding. Many times an intense paleness, of sudden appearance, accompanied by tachycardia, arterial hypotension, hypothermia, profuse sweating and facies similar to the Hippocratic, is what infers to us.

diagnosing internal bleeding and applying surgical treatment in time to save a life.

Paleness due to circulatory cause

Finally, there is pallor due to vascular or cardiac causes, dependent on changes in the caliber of the blood vessels in the skin or on special lesions of the aortic sigmoid valves. In it, the red color of the blood may remain normal, with the paleness originating from an emptying of the capillary networks of the dermis, either due to peripheral vasoconstriction or due to an abnormal reflux of blood to the left ventricle during diastole, a consequence of insufficiency of the aortic valves, as occurs in aortic insufficiency.

Pallor due to vascular causes may be due to vasoconstriction and obstruction. Due to vasoconstriction it can be observed temporarily: in emotions (emotional pallor); in the dizziness and vertigo that accompany nausea and vomiting; in intense pain or in cases of ingestion of some vasoconstrictor medications such as adrenaline, for example. In high blood pressure, arteriosclerosis and chronic nephritis, pallor due to vasoconstriction can also be observed, although more lasting, which is due to the angiospastic disorders that accompany these pathological processes. Paleness can be observed due to obstruction: in arterial thromboses and embolisms that are intense, painful and localized.

RUBICUNDE

Concept

In ruddy skin, the color of the skin is abnormally red or reddish, the complete opposite of paleness, to whose pathogenic mechanism all the factors of the paleness mechanism correspond, although acting, as is understandable, in an opposite

Semiognesis or pathophysiology

Ruddiness will occur whenever the thickness of the superficial layers of the epidermis is decreased or its transparency increased; when the blood vessels of the dermis are very numerous and thicker, or are full of blood, and, finally, when the red color of the blood increases, due to containing a greater proportion of hemoglobin or red blood cells or both at the same time.

Semiography and semidiagnosis

Ruddiness can be generalized and circumscribed or regional, and it can occur permanently or temporarily. When the skin is compressed, with a slides, for example, the redness disappears, which is easily understood when taking into account that compression empties the dermal capillaries.

Generalized redness

1. There is a congenital or familial redness that corresponds to the constitutional or congenital pallor that we mentioned before, and which is especially observed on the face and cheeks of individuals with very thin and transparent skin with abundant blood supply to the dermis. It is like constitutional and familial congenital pallor, and can be transmitted by inheritance.
2. Just as in anemias, the decrease in blood hemoglobin leads to paleness, in cases where the number of red blood cells and blood hemoglobin are increased, ruddyness will appear. This is the case of erythremia or polycythemia vera or Vaquez-Osler disease, in which hyperglobulia or an increase in the number of red blood cells per cubic millimeter is observed; Sometimes twelve to fourteen million red blood cells per cubic millimeter are observed. This is accompanied by red skin color, almost always with a special bluish or cyanotic tint.

3. Due to vascular causes, there is redness due to vasodilation, such as:

   a) Redness, emotional, nervous, which is none other than the blush that accompanies certain emotional states, more intense and frequent in some individuals than in others. It is mainly seen on the face, although it can also be seen in other regions of the body. It recognizes the same origin as toxic redness, that is, peripheral vasodilation, which by increasing the caliber of the blood vessels in the skin causes it to have a more intense red color.

   b) Toxic redness is observed after inhalation or ingestion of certain medications such as amyl nitrite, belladonna or its active ingredient, atropine. It is due to the peripheral vasodilation that these drugs produce and, like it, it is fleeting, transient and short-lived.

   c) Finally, red discoloration can also be observed in the skin and other regions that are exposed to the air and the sun (solar erythema), in fever, and in alcoholics where the ruddy discoloration of the face

   It achieves great semiological value, considerably reinforced by the appearance, especially on the cheeks, of a large number of dilated veins.

Circumscribed or regional rubicundity

It is one of the obligatory accompaniments of inflammation, which, as was known in the past, is reflected by a symptom.

drome in which the following four cardinal signs stand out: blushing, tumor, heat and pain. The redness is explained in these cases by the vasodilation of the dermal vessels, filled with blood, as a consequence of the great circulatory activity that the inflammatory process determines.

Circumscribed or regional redness may also be due to a local mechanism of vasodilation due to allergic processes.

CYANOSIS

Concept

Cyanosis (from the Greek kyanos: blue) is called the bluish, blue or violet color of the skin, mucous membranes and organs due to the profound increase in hemoglobin (Hb) reduced in the blood above 5 g per 100 mL, or due to the existence in it of other stable derivatives of hemoglobin such as: methemoglobin and sulfhemoglobin.

Semiognesis or pathophysiology

From the definition itself we understand:

1. That cyanosis cannot appear in those who have less than 5 g (33%) of Hb per 100 mL of blood.
2. That there will be two fundamental types of them:

a) First group: those produced by accumulation of more than 5 g of reduced Hb per 100 mL of blood.

b) Second group: those produced by the presence in the blood of a stable derivative of Hb; The latter constitute toxic cyanosis.

Cyanosis of the first group

In turn, they can be motivated by different mechanisms. In fact, there can be more than 5 g of reduced Hb per 100 mL of blood, due to:

1. That the blood remains in the tissues longer than it normally should.
2. That the blood leaving the heart has a higher level of reduced Hb than normal.
3. That the two previous factors coincide in the same case.

1. That the blood remains in the tissues longer than it normally should. This motivates a longer and greater exchange of oxygen - which the blood gives to the tissues - and carbon dioxide - which the tissues give to the blood - that is longer and greater than usual and which is manifested in the end by an increase in the concentration of reduced Hb in that capillary blood, which rises above 5g% (threshold figure for cyanosis). These are the so-called peripheral cyanosis.

The greater permanence of blood in the tissues is produced by:

a) Stagnation of blood in the tissues, which may be secondary to a general cause, such as a decrease in impulsive force due to heart failure, especially right ventricular failure; secondary to a local cause, such as occlusion of one or more venous trunks or poor drainage of the venous circulation of a given territory, due to intrinsic inflammatory or tumor processes of the vein or extrinsic compression of the vein, due to neighboring tumor or scar processes.

b) Abnormal blood distribution in the extremities, with decreased arterial blood supply and venous reflux.

2. That the blood at its exit from the heart already carries a higher level of reduced Hb than it normally should have. Under these conditions, if the blood reaches the tissues with a reduced Hb level greater than normal, when carbon dioxide passes through it at that level, even if it is done in a normal proportion and time, it is possible that this figure, together with that previously brought by the blood from the heart, produces a total greater than 5 g of reduced Hb per 100 mL of blood and cyanosis appears. This type of cyanosis, which is due to the blood leaving the left heart already altered in its chemical composition, is called central cyanosis.

The blood will leave the left ventricle with a reduced Hb level greater than normal, fundamentally due to two mechanisms:

a) Pulmonary mechanisms are those due to a lack of oxygenation of the blood in the lungs and may be due to a lack of oxygen in the inspired air, even if the respiratory system is normal. For example: great heights or depths and, in general, rarefied environments (submarine, tunnel, mine); or normal level or tension of oxygen in the inspired air, but poor condition of the respiratory system caused by:

Poor ventilation, that is, because a sufficient amount of inspired air does not reach the alveolus and a sufficient amount of alveolar air that has already undergone its gas exchange with the blood is not renewed.

Poor diffusion of oxygen from the alveolus to the capillary (alveolocapillary block).

Decrease in the active breathing surface of the lung tissue, as occurs in large pulmonary condensations that replace aerated lung tissue with solid tissue, or in large pulmonary fibrosis, etc.

b) Circulatory mechanisms, fundamentally reduced to abnormal communication (short circuit) between the right or left chambers of the heart or between the thick vessels that emerge from it, as long as there is sufficient blood flow from the right chambers and the pulmonary artery to the left chambers and aorta artery, because the abnormal pressures that the congenital disease creates in it determine this.

3. That the two previous factors coincide in the same case, that is, the factors that determine the types of peripheral and central cyanosis. As occurs in heart failure, in which a higher concentration of reduced Hb is simultaneously added to the peripheral circulatory stasis that it determines when blood leaves the left ventricle, due to poor pulmonary oxygenation motivated by the presence, to a greater or lesser degree, of the three factors that we have just mentioned, as pulmonary causes of cyanosis. This type of cyanosis is called: mixed cyanosis.

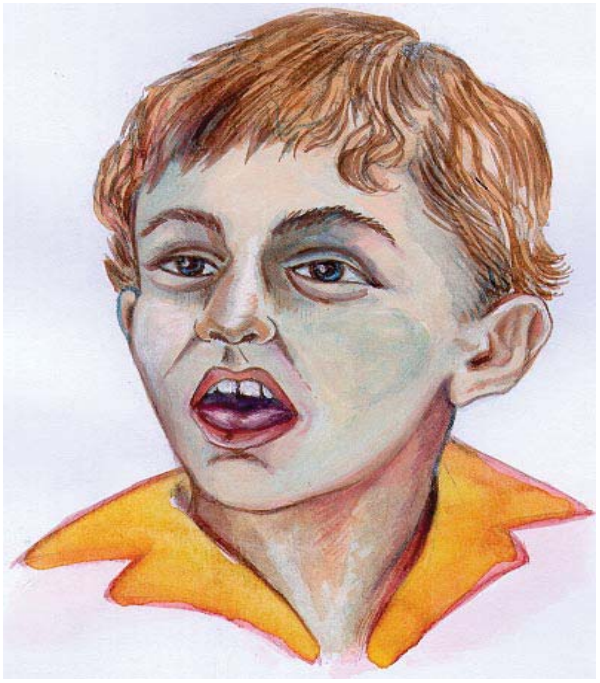


Fig. 25.17 Cyanosis due to methemoglobinemia.

Cyanosis of the second group

Cyanosis produced by the presence of a stable hemoglobin derivative (toxic cyanosis). The presence of methemoglobin or sulfhemoglobin makes blood oxygenation in the lung difficult, since these hemoglobin derivatives are not easily dissociable, having lost their affinity for oxygen. They give a leaden bluish color. Methemoglobin produces cyanosis when it reaches 20% of the total hemoglobin in the blood. There are familial cases, but cyanosis is generally due to exogenous toxins (nitrites, phenacetin, sulfanilamide, anilines) that penetrate through ingestion, inhalation or through the skin. Enterogenous cyanosis is due to the transformation of nitrates into nitrites in the intestine by bacterial action. The spectroscopic study of blood reveals these bodies.

Semiotechnics and semiography

ntensity

Cyanosis can be light or intense, and its color varies from a barely perceptible blue to a dark blue, almost black.

Location

The bluish color of cyanosis, both in the skin and mucous membranes, is usually diffuse, but may be limited to one region of the body. There is cyanosis generalized to the entire body or localized to a part or region of it.

Cyanosis (fig. 25.17) becomes more visible in certain regions that favor its appearance, such as the lips, nose, cheeks, auricles, hands and feet, especially the tips of the fingers and the nail bed. Therefore, these regions will never cease to be carefully inspected, when we look for cyanosis, especially in its mild forms, in which it is not always easy to decide if it exists. It is explained that the bluish color is more visible in them because they are distal regions, that is, located at a greater distance from the heart, because their epidermis is thinner and their dermis more vascularized, all conditions that, as is well understood, converge in facilitating the appearance of the characteristic color of cyanosis.

When studying a case of cyanosis, the mucosa of the lips, tongue, palatine vault and pharynx should also be carefully inspected.

Even though the bluish color of deep organs, such as the liver, spleen, kidneys, etc., cannot usually be appreciated, because these viscera are not visible, so it lacks semiological interest, however, in circumstances that make its inspection possible, such as during surgical operations, it acquires extraordinary semiological value, since it denounces the existence of some anesthesia accident, among which, due to its severity, respiratory syncope stands out, especially if not there is tracheal intubation. Cyanosis of the deep organs is, in this case, the alarm signal that puts the surgeon, more than once, in a position to save the life of his patient, compromised by the respiratory disorder responsible for the observed discoloration.

Slavine cyanosis affects the head, neck and upper extremities, and is observed in mediastinal syndromes that compress the superior vena cava.

Finally, we must note, as a complement to this general clinical study of cyanosis, that its intense and prolonged forms are usually accompanied by a special deformity of the last phalanx of the fingers and toes, especially the first ones, which we will study in respiratory semiology and which are called hippocratic or drumstick fingers.

Diagnosis

The positive diagnosis of cyanosis is made by checking the more or less bluish color of the skin, mucous membranes and organs.

The differential diagnosis with other similar skin discolorations can be made by trying to eliminate the blood by means of compression or vitropressure on the area of discoloration suspected of cyanosis and seeing if the discoloration disappears or is attenuated by reducing the amount of blood locally in the area being explored. If the discoloration persists, despite having reduced the blood supply to that area, it should not be cyanosis. However, sometimes it will be difficult to clarify the diagnosis clinically and in that case the assessment of reduced Hb in arterial blood will definitively make the diagnosis by verifying the presence or not, in it, of 5g or more, of reduced Hb per 100 mL of blood.

The diagnosis of toxic cyanosis will be made by verifying the abnormal variety of circulating hemoglobin by spectrophotometry.

The diagnosis of the variety or type of cyanosis, central or peripheral, will be made taking into account that: central cyanosis is commonly generalized to the entire body and is not modified because the cyanotic extremity is raised above the plane of the heart to facilitate its emptying, which favors the return of venous blood to the heart; In addition, the skin temperature is maintained:
It is a warm cyanosis. Peripheral cyanosis will usually be limited to one region of the body; The skin in that region will have a decreased local temperature, cold cyanosis, and when venous return is favored, through the maneuver indicated above, it will disappear, as the cause (blood stasis) that produces it ceases.

Semidiagnosis

Toxic cyanosis (see fig 25 17)

They are observed in people who ingest some compound capable of producing them: nitrites, chlorates, acetanilide, phenacetin, sulfonal, trional, etc. Mentioned here are the so-called enterogenic cyanosis due to the production of methemoglobin from intestinal poisoning.

Peripheral cyanosis

They are observed:

1. Due to stasis or non-organic stagnation:

Both in heart failure, specifically the right ventricular type, although commonly this cyanosis will be of a mixed type, as we said before, and in the syndrome of venous circulatory insufficiency of the lower extremities, especially due to varicose veins.

2. Due to stasis or stagnation of organic cause:

Thrombophlebitis, compression of venous trunks by neighboring tumors in the pelvis, groin, axilla, mediastinum, neck; or by neighboring scar tissue, due to radiation or surgery, in those same regions, from tumors or lymph node masses.

3. Due to abnormal distribution of blood in the extremities:

a) Functional: Raynaud's disease, acrocyanosis.

b) Organic: acute or chronic arterial occlusion with venous reflux.

Central cyanosis

They can be:

1. Due to pulmonary causes:

a) With integrity of the respiratory system, in the disease of high altitudes or mountains (Andean disease), or in workers in poorly ventilated tunnels and mines.

b) Those produced by alterations of the respiratory system can be (fig. 25.18):

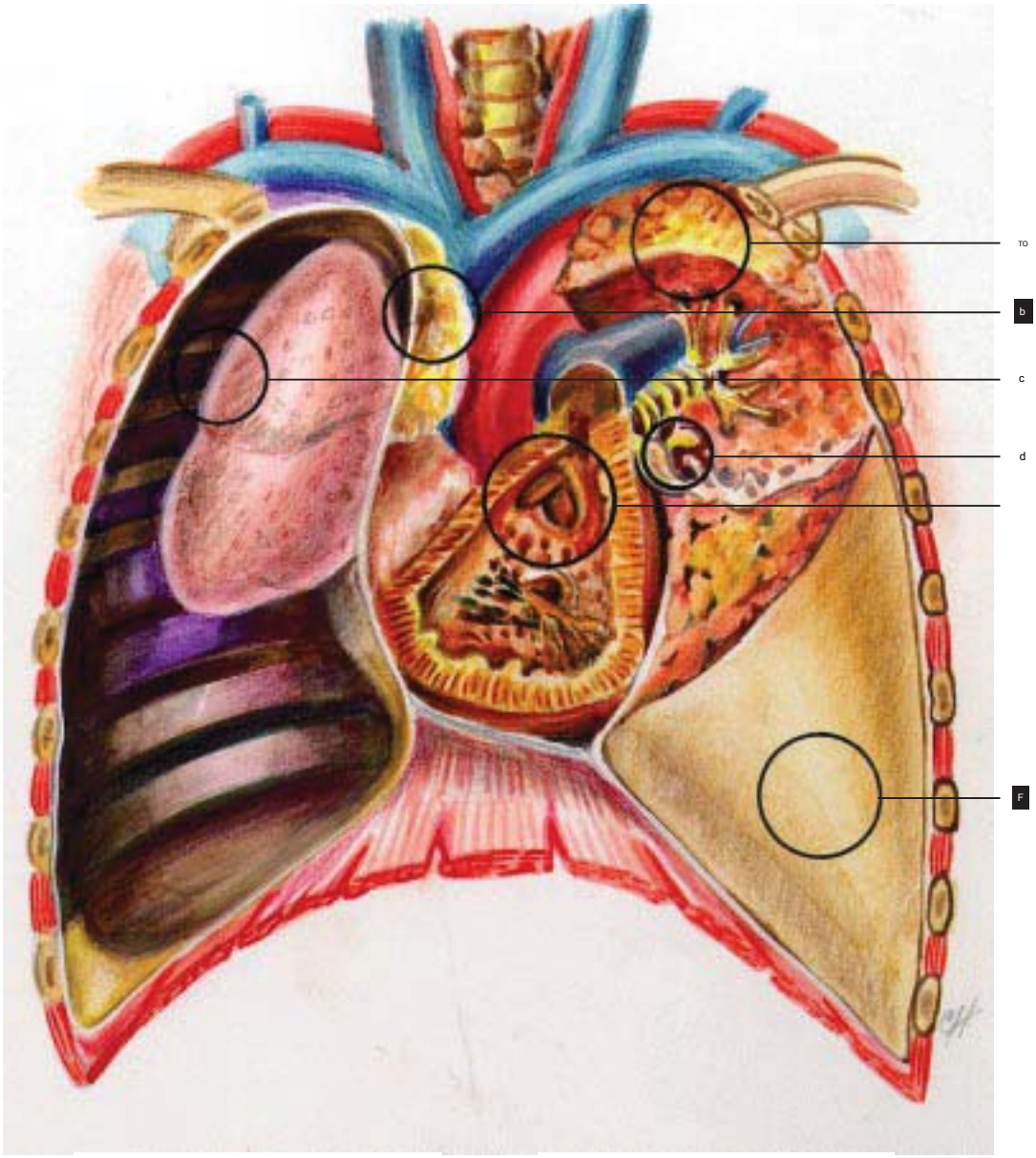
Due to poor ventilation: functional, as in bronchial asthma, and organic as in laryngitis, bronchioalveolitis or extrinsic compression of the larynx, trachea or bronchi due to neighboring tumors.

Due to alveolocapillary blockage: pulmonary interstitial fibrosis and primitive or secondary sclerosis of the pulmonary arterial capillaries.

Due to reduction of the respiratory area: massive pneumonia, confluent bronchopneumonia, bronchiectasis with a large peribronchial fibrous reaction, large pleural hemorrhages or large pneumothoraces, which compress the lung, etc.

2. Due to circulatory causes:

a) Congenital abnormal communication, between atria or ventricles, or univentricular heart, or in the communication between aorta and pulmonary arteries, or several of them at the same time; or congenital malposition of an arterial trunk, for example: aorta protruding from the right ventricle or riding on the interventricular septum.



AN MON A LOB LAR Partial hepatization of the lung causes pulmonary hypoventilation and raises the blood concentration of reduced hemoglobin

BT MOR SD LM D AST NO Mediastinal tumors that compress the superior vena cava are characterized by cyanosis, edema and dilation of the veins of the face and neck, and exolphthalmia.

CN MOT RA n some patients pneumothorax by reducing lung ventilation is accompanied by cyanosis

DC RP0S TRA OS L0s Foreign bodies lodged in the trachea and bronchi, hinder lung ventilation and reduce oxygen pressure in the alveoli

T TRALOGAD FALLOT l venous-arterial shunt due to ventricular septal defect and decreased blood flow in the lungs, reducing blood oxygenation

FD RRAM PL RAL Compression of part of the lung due to pleural effusion decreases pulmonary ventilation and blood oxygenation

Fig. 25.18 Semidiagnosis of central cyanosis.

Cyanosis initially allows these heart diseases to be classified into: acyanotic and cyanotic, a fundamental basis for their clinical study. Mixed cyanosis is typical of congestive heart failure.

YELLOW COLORATION

We frequently observe in the clinic the existence of a yellow or yellowish discoloration of the skin and mucous membranes. This is due, most of the time, to an increase in circulating bilirubin; On other occasions, much rarer, the yellow coloration is due to the impregnation of the external integument by pigments of a nature other than bilirubin, or by yellow chemical substances.

In the first case, the yellow coloring of the skin and mucous membranes is called jaundice, ictero (from the Greek icteros: yellowness), or jaundiced syndrome; In the second case, it is pseudoicterus or pseudoicterus, the name of which is meant to indicate that it is something that resembles true jaundice, but is not, and is therefore a false jaundice or pseudoicterus (pseudo: false). Among pseudoicteri we will study, mainly, the one that has its origin in an accumulation of carotine (provitamin A) in the body and the one that is due to the ingestion, often for simulating purposes, of picric acid, or derivatives of acridine used as therapeutics in some diseases.

Jaundice

Concept

Jaundice is called the yellow coloring of the skin, mucous membranes and organic fluids, which is due to their impregnation with bilirubin, abnormally increased in the blood plasma (hyperbilirubinemia).

Semiogenesis or pathophysiology

It is necessary to remember, even briefly, the fundamentals of bilirubin metabolism and hepatobiliary physiology.

Circulating human red blood cells are destroyed depending on their age (they last around 120 days) in the reticuloendothelial system.

The precise mechanism of catabolism of the hemoglobin molecule in the reticuloendothelial system is not completely known, however the first bile pigment formed is probably biliverdin, which is quickly reduced to bilirubin.

Bilirubin, which is relatively insoluble in water, is released from the reticuloendothelial system and passes into the plasma, where it binds to plasma proteins, predominantly albumin. There are other sources of origin of this bilirubin (liver and marrow). This bilirubin gives the indirect reaction with diazotized sulfanilic acid having therefore been called indirect bilirubin. At the level of the hepatocyte membrane, an uptake process occurs by virtue of which it is released from albumin. Two proteins present in the cytoplasm of the hepatocyte intervene in this uptake process, which have been called proteins Y and Z.

Once the bilirubin is captured, it is conjugated with glucuronic acid.

Schmidt and Folafont, using different methods, demonstrated that direct bilirubin was a glucuronide of bilirubin and that there are monoglucuronide and diglucuronide forms of it.

The diglucuronide form of bilirubin has the liver as the only source of formation in mammals, and is increased in biliary obstructions and liver damage.

The glucuronization of bilirubin is carried out in the liver, once the indirect bilirubin has been released from its protein, and this glucuronization is carried out at the level of the smooth endoplasmic reticulum of the liver cell by the activity of an enzyme called glucuronyltransferase.

Once bilirubin is conjugated in the liver cell with glucuronic acid to form bilirubin glucuronide, it is excreted through the bile ducts as bile (Table 25.1). Currently, unconjugated bilirubin is called bilirubin, which is found in plasma, and conjugated bilirubin, which is always found in bile under normal conditions.

Little is known about the excretory mechanism, although it is known that conjugation with glucuronic acid is necessary.

The excretory mechanism seems to be affected in certain liver processes such as cholestatic hepatitis and by the action of drugs such as methyltestosterone, etc.

Table .1

suitability	or play	Xcreci
BLOOD	EPATOCYTE	BILE
	Bilirubin	Conjugated
	+	bilirubin
	glucuronic acid	
Bilirubin	+	
	Glucuronyltransferase	Bilirubin
+		glucuronide
Protein	Bilirubin glucuronide	

This brief account of the metabolism of bilirubin allows us to conclude, in summary, that for circulating bilirubin to be maintained within its normal limits, the following are necessary:

1. That it is produced in a normal quantity by the reticuloendothelial cells that manufacture it at the expense of the hemoglobin released by the red blood cells in their physiological destruction process.

2. That the collection is carried out correctly.

3. That conjugation and transport take place in adequate proportion by the liver cell.

4. That it is excreted without obstacle through the bile, intrahepatic and extrahepatic ducts, pouring into the duodenum in normal

It will be easy for us to understand now that any pathological process that alters the action of these fundamental factors that intervene in maintaining normal bilirubinemia will be capable of giving rise to the appearance of jaundice, by raising the concentration of bilirubin in the blood

Hence, four main mechanisms causing jaundice can be distinguished:

1. Increase in the formation of free (unconjugated) bilirubin. The most frequent cause is the exaggerated destruction of red blood cells.
2. Disorders in the uptake of bilirubin at the level of the liver cell.
3. Disorders in the conjugation of bilirubin with glucuronic acid.
4. Difficulty in the excretion of conjugated bilirubin. This can occur within the liver cell or at the level of the intrahepaic or extrahepatic bile ducts.

Semiotechnics

The yellow color of jaundice depends essentially on the impregnation of the tissues by bile pigments accumulated in the blood. If the icteric skin is compressed with a slide, as we did in cases of cyanosis, the yellow coloration persists, and even becomes more evident, since it is sometimes partially hidden by the coloration given to the skin by the blood sheet of the dermis. The opposite happens than in cyanosis, which disappears due to vitro pressure. It is necessary, therefore, for bilirubin to pass into the tissues through the wall of the capillaries, which is permeable to the pigment, for the yellow color to appear. Furthermore, it is known that certain tissues have a great affinity for bilirubin, while others have almost no affinity for bilirubin, which is why they appear very little or not colored yellow at all. Among the first recipients of bilirubin are the skin, sclera and heart valves; Among the latter, the conea, muscles and nervous system are indicated.

semiography

We already said that jaundice was a syndrome, and the yellow color of the skin was one of the symptoms that make it up. We will now study the clinical characteristics of the jaundice yellow color and the most important symptoms that accompany it in the syndrome we study.

The intensity of the yellow color of the skin and mucous membranes is very variable, ranging from the barely visible yellow color of sub-jaundice to the clearly perceptible yellow color of so-called frank jaundice. Sometimes, the yellow color cannot be seen in the skin or mucous membranes, despite the fact that bilirubin is increased in the blood plasma. We then say that we are facing a latent jaundice. It is believed that the jaundiced yellow color begins to become visible when the bilirubin level in the blood is 1.5 mg/100 mL and is clearly noticeable when there is more than 2 mg of pigment in the same amount of blood. Thus, latent jaundices would be, in terms of the amount of blood bilirubin, above normal, without reaching the figure of 1.5 mg/100 mL, at which sub-jaundices would begin, with frank jaundices above 2 mg/100 mL.

Bilirubin has an affinity for elastic fibers rich in relaxin, hence the yellow color of jaundice reaches greater intensity and appears earlier in certain regions, which, therefore, should always be inspected with great care:

sclera; underside of the tongue; palatine vault, especially the soft palate; skin of the forehead, chest, abdomen and the inner surface of the upper and lower limbs.

The tone or tint of the yellow color is not uniform in all cases of jaundice, presenting, on the contrary, variations of great semiological interest. Jaundice with flavinic, rubinic, verdinic and melanic tinges are distinguished.

In flavine jaundice (from the Greek, flavus: yellow), the color is light yellow or lemon yellow, and is observed with great frequency in hematogenous or hemolytic jaundice.

Rubinic jaundice has a more intense yellow color, with a reddish tint (from which it owes its name) or brownish red. It generally manifests the existence of hepatogenic jaundice, due to alteration of the liver cell, that is, of the hepatocellular type. We can add that on some occasions this rubinic dye acquires a very characteristic reddish-orange color, such as in icterohemorrhagic leptospirosis.

Greenish jaundice is characterized by presenting a greenish color, more or less frank or greenish yellow, due to the presence of biliverdin, in a more or less large proportion in the circulating blood and tissues.
It occurs almost exclusively in obstructive jaundice.

Melanistic jaundice, dark in tone, sometimes almost blackish, occurs in long-lasting and highly intense obstructive jaundice, such as cancer of the head of the pancreas. It is possible that the dark hue is due to prolonged skin irritation from

Bile pigments lead to an increase in epidermal melanic pigment.

Among the skin symptoms that accompany yellowness, pruritus or itching stands out, reported by scratching marks that become very visible on the skin of some patients. This symptom is not constant and its intensity is highly variable. In some cases it becomes intolerable, even compromising the general condition of the patient, due to the lack of sleep it causes. It is felt, above all, in the interdigital spaces, armpit, groin, abdomen and extremities, and increases during the night, which explains the insomnia.

It is not well known what this is due to, having been attributed to bile salts, which irritate the skin, to choline, porphyrins or to substances with a chemical composition close to histamine.

In the inner corner of the upper eyelids of many jaundiced people, a yellowish, rounded, elliptical or oval plaque, slightly protruding, is observed, which is called xanthelasma. It is attributed to a local deposit of cholesterol, greatly increased in the blood of some patients with obstructive jaundice.

Some interesting digestive symptoms make up the jaundice syndrome; Among them, the lack of appetite (anorexia) and the disgust for certain foods stand out, sometimes very marked, constipation and, above all, the discoloration of fecal matter, which can be complete - acholia - with the stool appearing whitish, like ash or putty, or, less marked, constituting what is called hypocolia. Obviously, it will be especially in mechanical or obstructive jaundice, where the discoloration of fecal matter will be observed, the more intense the more complete the existing obstruction in the bile ducts. On the contrary, in hemolytic jaundice, hypercolia is observed when the elimination of conjugated bilirubin and its derivatives (stercobilinogen and stercobilin) increases.

The urine of jaundiced patients has a light or dark brown color - choluria - and the foam it forms is a very visible yellow color, staining clothes yellow. In hemolytic jaundice, clothing is not stained and the stronger color that the urine takes is due to urobilinogen and not bilirubin, since in this type of jaundice unconjugated bilirubin predominates, which is insoluble in water and is not eliminated by the kidney.

Finally, we must mention the circulatory and nervous symptoms that can be observed in jaundice.
Among the former, we find bradycardia, of the sinus type, attributed to bile salts or choline: arterial hypotension, and, in severe forms of jaundice, nasal, gingival and subcutaneous hemorrhages due to the deficient formation of coagulation factors. Among the latter, the insomnia, already mentioned, and asthenia. Interesting visual disorders are observed in many jaundiced patients: hemeralopia and xanthopsia. Hemeralopia is translated by a more or less marked, sometimes total (night blindness) difficulty in seeing in the dark. It is attributed to defects in the formation of visual purpura due to vitamin A deficiency, which, as is known, is formed mainly in the liver at the expense of provitamin A or carotene. Xanthopsia consists of yellow vision of objects.

Semidiagnosis

1. Jaundice due to increased formation of free bilirubin (hemolytic jaundice).
2. Jaundice due to disorders in the hepatic uptake of bilirubin (Gilbert's disease and due to the action of some drugs).
3. Jaundice due to conjugation disorders:
   a) Enzymatic immaturity (newborn jaundice).
   b) Absence of glucuronyltransferase (Crigler-Najar disease).
   c) Inhibition of glucuroconjugation (jaundice due to breast milk and drugs).
4. Jaundice due to excretion disorders:
   a) Intrahepatic cholestasis (hepatitis, cirrhosis, drugs).
   b) Extrahepatic cholestasis (stones, neoplasms).

Pseudojaundice

Concept

We already said that, in some cases, the yellow color of the skin could be due not to bilirubin, but to yellow chemical substances, such as picric acid, or to pigments of a different nature from bilirubin, carotene, for example, and that, then we say that there is false jaundice, that is, pseudo-jaundice.

Semiogenesis, semiography and semidiagnosis

We must now briefly consider the two main types of non-icteric yellowness: that which is due to an excess of carotene and that which depends on the accidental or voluntary ingestion of picric acid or other substances.

Carotine pseudoicterus is observed in individuals who predominantly or preferentially use certain vegetables rich in carotene in their diet, such as carrots, or who abuse the consumption of eggs. The yellow color they present is due to excess carotene ingested, to which is often added a functional liver deficit that reduces the formation of vitamin A at the expense of that pigment (provitamin A). This yellow color of the skin is frequently observed

cia in diabetics and in patients with hypothyroidism.
In these cases, the palms of the hands, soles of the feet, cheeks and forehead are stained more intensely;
but not the sclerae, the soft palate, or the urine, which differentiates it from true jaundice. The normality of bilirubinemia and the absence of bile pigments in the urine can also be verified, to which are added the history of the case, showing the consumption of foods rich in carotene.

Picric pseudojaundice was seen a lot during wars. Many malingerers used the ingestion of picric acid as a means to make people believe that they had real jaundice and to be excluded from military service. The differential diagnosis rests here, as in the previous case, on the absence of the cardinal symptoms of true jaundice and, above all, on the demonstration of the existence of picric acid in the urine. It can also be produced by phenolphthalein and acridine derivatives, and in uremia by urochrome.

TAN OR DARK SKIN COLORATION MELANODERMA

Concept

Frequently, and for various reasons, it is observed that the skin takes on a dark color, more or less intense, almost blackish in some cases. It is said then that there is a melanoderma (from the Greek melanos: black; derma: skin).

Semiognesis and semiography

Melanoderma is a pathological alteration of skin color and depends on changes or modifications in the quantity and distribution of the second of the main factors that we point out as responsible for normal skin color: nelanin or black coloring matter contained in the cells of the deep epidermal layer.

The amount and distribution of melanin in the cells of the basal layer and stratum malpighian of the epidermis undergo important variations in the normal state, and are responsible, as we have already said, for the different coloration of the skin in human races, in different individuals of the same race and in different regions of the same individual. The melanic pigment also increases in all those regions of the skin exposed to the action of solar rays, heat, compression, or other physical or chemical agents, this special way that the skin has to react is very peculiar, becoming pigmented and acquiring, as a consequence, a darker color in the irritated areas. It is not strange, since in various pathological conditions dark discolorations are also observed.

skin rashes dependent on alterations in epidermal melanin. The dark coloration of the skin, characteristic of melanoderma, can be localized or circumscribed, or generalized in more or less extensive areas.

Semidiagnosis

Moles or nevi, of congenital origin, and ephelides or freckles are circumscribed pigment spots, circular in shape and yellowish or light brown in color, which appear under the action of the sun in individuals with very white skin and blonde or red hair and which have no special pathological significance, the same as chloasma (from the Greek cloazo: spot) or mask of pregnancy, consisting of dark pigmentation, of intense variable and diffuse, which appears on the face, during pregnancy, especially on the cheeks and forehead, accompanying the greater pigmentation of the skin regions normally richer in melanin (nipples, areola, abdominal white line, etc.), which is also observed in pregnant women.

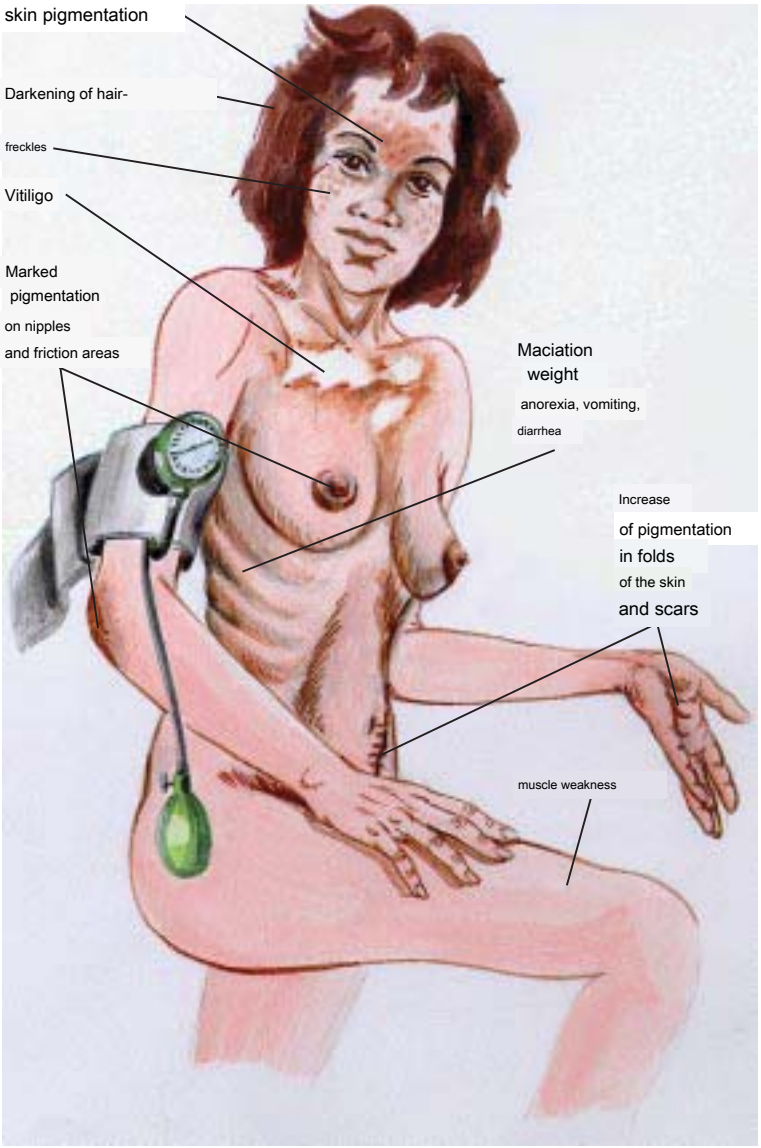
More important are the dark pigment spots left by some syphilitic papules of the secondary period (syphilides nigricans) and the numerous and widespread pigment spots seen in individuals who have suffered, or suffer, pruritic skin conditions, especially parasitic ones, such as ptyriasis (body lice), common in people with poor hygiene, in whom it is observed that almost the entire skin becomes stained, with a special pigmentation on the back, in the interscapular region, where the action of the parasites is most intense. Of great diagnostic importance is diffuse melanoderma, which is observed in Addison's disease (Fig. 25.19), frequently caused by fibrocaseous tuberculosis of the adrenal capsules or by autoimmune mechanisms. In it, a dark coloration of the skin is observed, more marked in the areas exposed to sunlight, pressure and friction such as: face, neck, back of the hands, forearms, waist and the skin that rests on the bony eminences. The mamelons, armpits, vulva and anus also appear very pigmented, while the palm of the hand appears almost free of pigment.

The mucous membranes also become pigmented, and it is very characteristic that what is observed on the free edge and inner surface of the lips, gums, soft palate and inner side of the cheeks.

In neurofibromatosis or Recklinghausen's disease, the characteristic skin and nerve trunk tumors are accompanied by diffuse melanoderma or patches of coffee-au-lait color.

In other cases, the rather tan-like dark coloration of the skin is not due to increased pigment.

Fig. 25.19 Melanoderma: Addison's disease.



melanistic as in the processes we have just mentioned. Tan pigmentation now depends on hemosiderin, an iron-rich substance that appears when there is intense destruction of red blood cells, and is observed in tan diabetes (hemochromatosis), characterized, in addition to the special pigmentation that gives it its name, by the existence of glycosuria and liver cirrhosis.

OTHER ALTERATIONS OF SKIN COLOR

It is interesting to study here, preferably, those alterations in skin color that are due to the lack or absence of the melanic pigment, or to its displacement from a certain area of the skin and concentration in the periphery of the same.

In albinism (from the Latin albus: white) the melanic pigment is missing, the skin has a very intense white color and the hair is a very pale blonde, almost white as well, which, with the bluish color of the eyes, gives these subjects an unmistakable appearance. There are cases in which albinism is not generalized, but rather occurs in a circumscribed area, such as achromic spots.
It is partial albinism which, as Padilla says, graphically, is nothing other than the inverse condition of the mole.

In vitiligo (from the Latin vitiligo: white spot), the melanic pigment moves towards the periphery, leaving a central area of milky white or matte white, surrounded by an area of increased, darker, very characteristic pigmentation. It can be located anywhere and is generally symmetrical; in many cases it extends to the entire body. Its cause is not well known,

even though it is attributed a relationship with neurovegetative disorders; It currently tends to be considered an immunological condition.

Achromic spots can be observed (from the Greek a: privative particle; chromes: color), after localized skin conditions, wounds, burns, etc. It is always interesting to study the sensitivity of these achromic spots, since, when the existence of thermal and painful anesthesia is verified in them, it should be suspected that they are of leprous etiology.

SKIN WETNESS

We must not forget the study of the moisture of the skin when it is examined during the general inspection of the patient, since both its abnormal dryness and its tendency to increased sweating are of great diagnostic value in many pathological states.

The humidity of the skin is a function, in large part, of the activity of the sweat glands, that is, of sweating. Normally, drier skin is observed under the action of the air and the outside cold, while the heat causes more or less abundant sweating as a defensive reaction.

In a pathological state, sweat secretion may be increased, constituting what is called hyperhidrosis (from the Greek hyper: increase or excess; hydro: water); or decreased, hypohidrosis; or absent, anhidrosis.

IPER IDROSIS

Hyperhidrosis, diffuse or generalized, occurs in various morbid processes, especially in infectious diseases. It is also found, with great intensity, accompanying the critical defervescence of many hyperthermias.

In pulmonary tuberculosis, especially in its excavated fibrocaseous forms, abundant sweating is observed during the night or in the early morning, with great weakness, which is very characteristic and is described as hectic sweats or night sweats of consumptives. They coincide or not with the drop in temperature and seem to depend on tuberculous toxemia.

Of great diagnostic interest is the hyperhidrosis of states of peripheral vascular collapse, very characteristic, which is distinguished from the previous ones by being accompanied by an extraordinary coldness of the skin (batrachian skin or frog skin) and by its special viscosity, which gives the palpating hand an unmistakable sensation of cold sticky moisture. These conditions have a serious prognosis.

Patients with kidney diseases, chronic nephritis, for example, also present hyperhidrosis. Profuse sweating is, in this case, an effort by the organism to release retained excretory substances, thus compensating for the existing deficit in renal elimination.

Other times, increased sweating is limited to a certain region. Thus, in vagotonic subjects, described by Eppinger and Hess, and to whom we have already referred, a particularly abundant axillary sweating is observed, which slides in large drops visible on the lateral wall of the thorax. Likewise, we can mention here the sweating of the hands that these patients also present.

IPO IDR0SIS

The decrease in sweating or hypohidrosis with abnormal dryness of the skin is observed in all those pathological processes with great dehydration, in which large amounts of water are eliminated, such as profuse diarrhea accompanied by vomiting, chronic nephritis with great polyuria and sugary diabetes or insipidus. It can also be observed when the absorption of liquid through the intestine is decreased.

ANIDROSIS

The complete absence of sweat secretion or anhidrosis may occur in the processes responsible for hypohidrosis when they reach great intensity; But rather, anhidrosis is observed limited to certain skin areas in which, due to pathological disorders, the sweat glands have been destroyed or paralysis of the synpathetic nerves that control them has occurred.

BROMIDROSIS

We say that bromidrosis exists (from the Greek bromos: bad smell), when the sweating presents a special, very characteristic stench. This annoying alteration of sweat secretion seems to be related to the elimination of fatty acids or ammonia derivatives through sweat, or to a fermentation suffered by it after having been secreted. It seems to be due to racial factors or individual constitution and is preferably observed in the armpits and feet, especially.

CHROMIDROSIS

Colored sweating or chromidrosis (from the Greek chromium: color) is very rarely observed. Some jaundiced patients present yellow-colored sweat, and red sweat may be seen near abscesses caused by the prodigious bacillus, just as bluish sweat may be seen near regions infected with the pyocyanic bacillus. Even though its existence is admitted by some authors, the observation of red blood sweat is more than exceptional, that is, the

hematidrosis. Outside of these cases, the claim that colored sweat occurs should be taken with great reserve, often being a trick, or lack of hygiene, or clothes that fade.

URIDROSIS

It is sweating highly loaded with urea that is observed, due to vicariant action, in severe renal failure, with great retention of urea and other nitrogenous products.
Etymologically, it precedes the Greek ouron: urine. In these cases, the urea and sodium chloride excreted when the water dries up are deposited on the skin in the form of a white powder called uremic frost.

VASCULAR SKIN LESIONS

CUT NEAS EMORRHAGIES

They are due to blood coagulation disorders or increased vascular permeability. Purpuras are skin bleeding in the form of spots that appear in trauma and in purpuric and hemorrhagic syndromes, receiving different names: petechiae, vibes, ecchymoses, hematomas. They are hemorrhagic suffusions, that is, they do not disappear when pressure or extension is exerted on the skin that covers them, as occurs with congestive phenomena and vascular dilation, such as erythema and telangiectasia.

A. Purpuric lesions.

1. Petechia: small, round, red or purple macule, the size of a dot, a pinhead, or a lentil (1-2 mm).

2. Vibes: they are small linear hemorrhages, in the form of threads a few millimeters long.
3. Ecchymoses: they are irregular or rounded spots, larger than the previous ones and whose color varies over time, depending on the state of reabsorption or evolution of the blood shed under the skin: purple, brown, yellow, green.

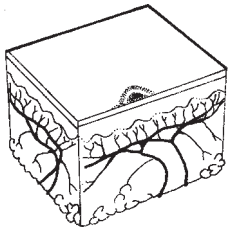
B. Hematoma.

Collection of extravasated blood, of even greater volume, that is prominent over the skin, in which a sensation of reluctance can be palpated in the first moments that is later followed by a sensation of hardness, observing in the skin that covers it, the color changes that the extravasated blood undergoes in its reabsorption process. Its size varies, according to the accumulation of blood.

OTHER VASCULAR SKIN ALTERATIONS

Strawberry angioma (Bouchard's red mole). Papulary round. Red or purple. Observed on the trunk and extremities. It can whiten with pressure. Normal age-related skin condition. Although it is also commonly observed in chronic liver diseases such as cirrhosis. It usually has no clinical significance (fig. 25.20).

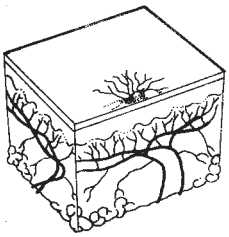
Fig. 25.20 Bouchard's red mole.



Telangiectasias (spider veins and star veins).
The term telangiectasia etymologically means dilation of the extreme vessels, that is, of the capillaries, both arterial and venous:

a) Vascular mite, called spider in English-speaking countries, and the only lesion that we have become accustomed to calling telangiectasia, is a lesion of the arteriole, red in color, that has a central body with radiating branches. When the center is pressed, the radiating branches disappear (fig. 25. 21). It is seen on the face, neck, arms and trunk. Rare below the waist.
It can whiten with pressure. Associated with liver diseases, pregnancy and vitamin B deficiency.

Fig. 25.21 Spider vein.



b) Venous stars are bluish or red lesions, permanent, that is, they do not disappear with pressure. They are secondary to dilation of superficial venous capillaries. The shape varies: spider-like (similar to vascular spiders) or linear (similar to snakes). They are seen on the legs and anterior thorax. Associated with states of increased venous pressure (varicosities) (fig. 25.22).

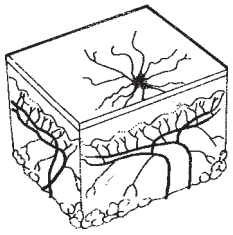


Fig. 25.22 Venous star.

COLLATERAL CIRCULATION

When there is an obstacle to blood circulation in the large arterial or venous vessels due to obstruction, compression or narrowing, all the vessels that anastomose with the affected trunk dilate considerably, in an effort to compensate for the existing circulatory difficulty. This vicariant vascular dilation, visible in the superficial vessels, which are what give it its extraordinary semiological value, and thanks to which the blood circulation interrupted by the obstacle is partially restored, is known in semiology by the name of collateral, arterial or venous circulation, depending on the obstructed vessel.

The semiological value of collateral circulation is very great. Through it, we know of the existence of deep vascular obstructions or compressions, which could go unnoticed if this valuable sign did not report them with great precocity and precision.

A distinction is made, as we said, between an arterial collateral circulation and a venous collateral circulation.

ARTERIAL COLLATERAL CIRCULATION

Arterial collateral circulation is observed, above all, in congenital stenosis of the isthmus of the aorta (aortic coarctation), a congenital malformation. Here a marked narrowing or constriction of the aorta is observed, frequently located after the birth of the left subclavian artery, at the same point where the ductus arteriosus empties into fetal life.

As a consequence, and to compensate for the obstacle and ensure adequate circulation in the lower regions of the body, extensive arterial collateral circulation develops between the arteries located above and below the narrowed aortic area. The intercostal, internal mammary, scapular and other arteries increase considerably in size, making their pulsations visible along their path where they can also be easily palpated.

VENOUS COLLATERAL CIRCULATION

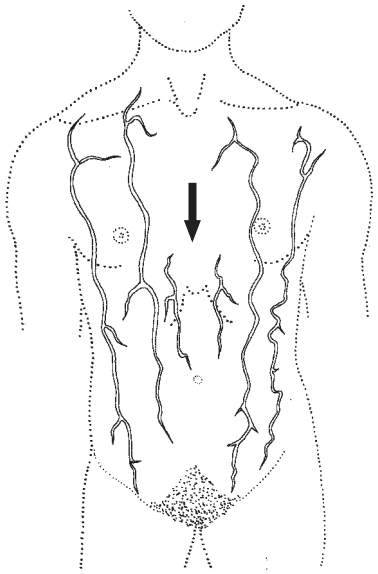
Much more frequent and, therefore, of greater semiological interest; It is presented to us in practice adopting one of the following main clinical types:

-Superior cava type venous circulation.
-Inferior cava type venous circulation.
-Portal type venous circulation.
-Mixed or portocaval type venous circulation.

Superior cava type venous circulation

It is observed when there is a circulatory obstacle in the superior vena cava, almost always due to constant compression. secondary to fibrous mediastinitis, a mediastinal tumor or an aortic aneurysm. It joins edema and cyanosis in esclavina to integrate a very characteristic and expressive syndrome. It is translated by a great development of the superficial venous networks of the upper part of the thorax, marking, on the sides of the thorax and the abdomen, a large venous trunk that runs from top to bottom towards the territory of the inferior vena cava, constituting the long superficial thoracoepigastric vein, which is enormously dilated and which, with other superficial and deep collateral branches, diverts to the inferior vena cava the return venous circulation that cannot drain through the superior cava. If we study the direction in which blood circulation takes place in this large dilated venous trunk by emptying a segment of it between two fingers, and raising one and the other of these alternately, to determine when the emptied section is filled with the greatest speed, we will find that the direction of the blood flow is from top to bottom, from the superior cava to the inferior (fig. 25.23).

Fig. 25.23 Superior venous cava type circulation.



Inferior cava type venous circulation

It will be found whenever there is an obstruction (thrombosis) or compression (abdominal tumor, large ascites) of the inferior vena cava. A marked development of the superficial venous networks is observed in the entire lower half of the abdomen, and as in the previous type, the long superficial thoracoepigastric vein is very dilated. It is also visible along the thorax and abdomen, only in this case, when studying the direction of the blood flow by the maneuver previously indicated, it will be found that it is carried out from the bottom up, diverting towards the superior vena cava. circulation

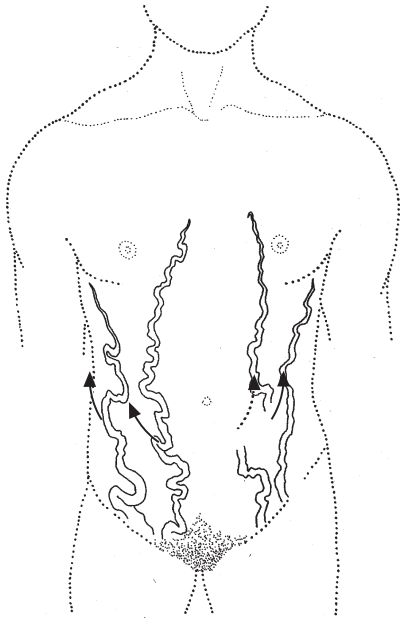


Fig. 25.24 Inferior cava type venous circulation.

venous vein stopped in the territory of the obstructed inferior cava (fig. 25.24).

Portal venous circulation

It occurs when the obstacle lies in the portal vein, or in its intrahepatic branches. It is either a portal obstruction, due to inflammatory thrombosis (pylephlebitis), or a tumor compression (nodal tumors of the hepatic hilum, for example), or an imprisonment of its intrahepatic branches by the sclerotic tissue of liver cirrhosis.

Collateral circulation is through the umbilical venous system, made up of the umbilical vein, which is not always obliterated, the paraumbilical veins and other accessory carrying systems. Upon inspection, a visible supraumbilical or lower thoracic venous network is observed, in which blood circulation is from bottom to top seeking the drainage of venous blood from the portal territory through the superior vena cava.

Characteristic of this type of collateral venous circulation, when it is pure, is the absence of venous dilations in the lower half of the abdomen, which distinguishes it from the inferior cava type or the portocaval type (fig. 25.25).

In some cases, the collateral venous circulation is accentuated around the navel, where it adopts a more or less radiated shape, with its radii represented by undulating veins.

This interesting variety of portal collateral circulation has been called the medusa head (fig. 25.26).

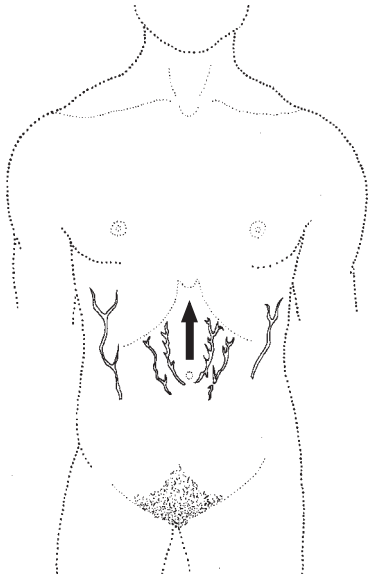
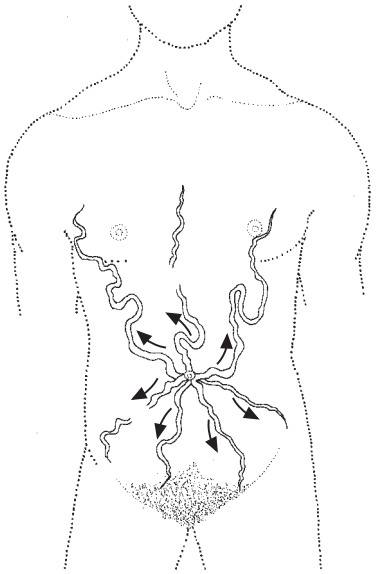


Fig. 25.25 Portal venous circulation.

Fig. 25.26 Portal venous circulation in the head of a jellyfish.



Portocaval type venous circulation

It is a type of mixed collateral venous circulation in which there is a circulatory obstacle in both the portal vein and the inferior vena cava. It is commonly observed in cirrhosis with ascites, as cirrhosis is determined by portal obstruction and ascites by compression of the inferior vena cava. Clinically, we observe a combination of both types of collateral circulation that identifies it, accompanied by the dilation of the superficial veins of the lower half of the abdomen and the long superficial thoracoepigastric vein, always observed.

pre that there is an obstacle to circulation in the inferior vena cava.

There are many other skin lesions that we will not discuss here, since they will be studied in the subject of Dermatology.

ALTERATIONS OF THE FANERA

HAIRS

Its semiological study is done through inspection and palpation, trying to establish the variations, both in normal and pathological states, that may present its characteristics, which are the following: quantity, distribution, implantation and quality, which translate into its appearance, color, length, thickness, resistance and trophic state in general.

The examination of the hair and its physiological variations were studied in Chapter 3 of Section I. The pathological alterations will be treated in Volume 2, in the chapter corresponding to the endocrine system.

U AS

In Chapter 3 of Section I, the concept, the examination guide or semitechnics, the physiological variations were established, and some definitions of nail alterations were provided.

We remind you that you must study their shape, appearance, resistance, growth and color, which can present alterations in the most diverse internal diseases, particularly those of a metabolic, infectious, neurovegetative nature, etc.

As we already studied, the normal nail grows around 0.5 mm per week: it is pink in color, with a smooth surface and elastic consistency. The whitish lunula occupies one fifth of its surface (fig. 25.27).

In anemia, the nails become pale; In polyglobulia they present a dark red color, and in cyanosis, bluish.

Its development is affected by diseases that compromise the general state (nutritional deficiencies, endocrinopathies, tumors, infections, circulatory disorders, etc.).

Below we will describe the types of nails that have the greatest clinical significance (see Fig. 25.27).

Terry nails. The nails appear white up to 1-2 mm from the distal edge where there is a normal pink area. The lunula may be darkened. Although Terry described these characteristics in liver cirrhosis, he observed that nail whiteness also occurred in congestive heart failure, diabetes, pulmonary tuberculosis, and rheumatoid arthritis.

Lindsay's Nails. The half and half nail. The proximal portion is whitish while the distal portion is red, pink or brown. The distal band comprises 20-60% of the nail. These nails are seen in chronic renal failure.

Mees lines. They are transverse white bands parallel to the lunula, which occur on the nail in the same relative position on each finger. They can be single or multiple. Although Mees described these changes in arsenic poisoning, similar white lines have been observed in acute and chronic renal failure, as well as in thallium poisoning, leprosy, malaria, psoriasis, heart failure, pellagra, Hodgkin's disease, pneumonia, heart attack, sickle cell disease, and infectious fevers. Drugs should be added to this list, especially cancer chemotherapeutic agents.

Muehrcke lines. They are two transverse white bands parallel to the lunula, which are observed in the nails of patients with serum albumin levels below 2.2 g/100 mL. Unfortunately, conditions that cause hypoalbuminemia often produce dull white nails, such as Terry nails in liver cirrhosis and half-and-half Lindsay nails in kidney failure.

Beau lines. They are transverse notches parallel to the lunula that occur on each nail in the same relative position. They are usually seen after serious infections or a severe period of illness.

Watch glass nails. Isolated or coexisting with clubbing toes, they are characterized by being convex, without the angle of less than 180° that is normally formed by the plane of the nail and that which passes through the dorsal surface of the nail phalanx (fig. 25.28). This type of nails looks

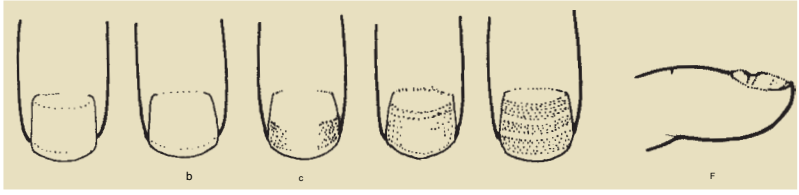


Fig. 25.27 Normal nail and pathological nails: a, normal; b, from Terry: c, from Lindsay; d, from Mees; e, from Muehrcke; f, from Beau.

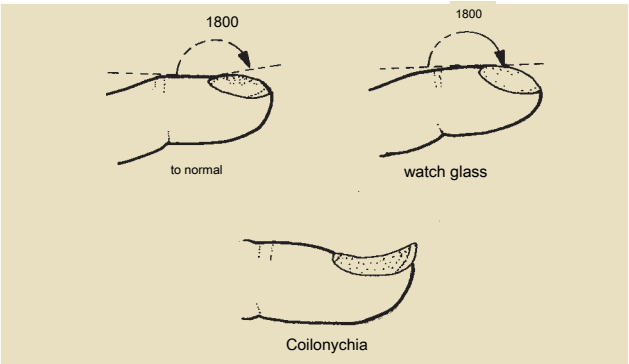


Fig. 25.28 Watch glass nail and koilonychia.

in lung cancer, bronchiectasis, chronic bronchitis, lung abscess, congenital heart disease with cyanosis and subacute endocarditis; sometimes also in ulcerative colitis, tumors of the digestive system and intestinal polyposis; There is a hereditary family form.

Excavated nails (koilonychia) or spoon nails (see Fig. 25.28). They are concave instead of convex and together with flattened nails (platonychia) they appear in anemia, and due to the use of strong soaps or detergents. Nail hypertrophy or thickening (pachyonychia) may be observed in tabes and polyneuritis.

The nails are sometimes hooked (onychogryphosis). In arterial insufficiency of the lower limbs, the nails thicken and often become brittle (onychorrhexis). In nephrotic syndrome and liver cirrhosis, the lunula of the thumb may disappear, which has been attributed to hypoalbuminemia. In pituitary, thyroid and parathyroid insufficiencies, the nails are atrophic, striated and fragile, with slow growth. Bitten nails (onychophagia) show states of anxiety and psychological tension.

26

Introduction

When performing this examination we must remember that adipose tissue is normally found there, so this is the moment in which we will observe if the patient maintains a normal weight or if he is thin because his panniculus has decreased or disappeared; or, on the contrary, if you are obese because you have increased your weight. Apart from the clinical impression obtained in the inspection, it is always advisable to verify the process using the scale or balance and compare it with the guiding tables on the normal average weights of each person according to their age, sex, height and constitutional type, such as those we have indicated in previous chapters, before definitively reaching conclusions regarding weight, especially in doubtful cases.

Likewise, it is necessary to complete the clinical examination of the patient before stating that the weight gain corresponds to obesity, since the subcutaneous cellular tissue may be increased because it contains substances that should not normally be found in it, or, as in subcutaneous edema, because it contains substances that are normally found in it, but in greater quantities than normal. With the previous reservation made, we will understand that any person whose current or real weight (the one obtained at the time of the exam) is 10% greater than their ideal weight (the one that corresponds to them according to the tables) is obese; and that any person whose current weight is 20% less than their ideal weight is thin.

EDEMA

CONCEPT

We say that there is edema (from the Greek oidena: tumor, swelling) when the interstitial fluid or plasma that occupies the intercellular or interstitial spaces is pathologically increased.

SEMIOGENESIS OR PHYSIOPATHOL0GY

The increase in interstitial or intercellular fluid is produced by an alteration of the mechanisms that normally regulate the distribution of water in the body. Consequently, the study of the pathophysiology of edema is not possible without reviewing these mechanisms.

The theory of the evolution of species in the development of life states that when primitive living forms moved from the sea,

where they are supposed to have originated, to the earth, they took with them a part of the sea that surrounded them, thus creating the first vital system, characterized by water inside the cell (intracellular water) and water outside it and around it (extracellular water), forming with the cell a biological unit and maintaining a constant physicochemical exchange between the cell and the environment.

As the living being evolved and went from the unicellular to the multicellular state and as the differentiation of tissues into organs and systems occurred, each one in charge of specific functions, it was necessary for the extracellular space or water to be able to reach all the cells and at the same time continue the physicochemical exchange with the environment, to be divided into two portions: one that would still remain in intimate contact with the cells: intercellular or interstitial water, and another that was within the vascular system: arteries, veins and lymphatics through which it could circulate and reach the most remote intercellular space and, in turn, come into contact with the environment and eliminate the final products of cellular metabolism from the body and obtain from it the nutritional elements essential for maintaining life: normally an adult ingests about 2,500 mL of water each day in the form of liquid and solid foods, which also contain water;

and eliminates the same amount distributed in 1,500 mL through the urine; 450 mL due to perspiration or perspiration; 450 mL through the lung and 100 mL through the feces. Thus the chemical composition of the extracellular water of today's human beings very closely resembles that of the seas of the geological era (Cambrian seas) in which life is supposed to have passed to land. Conceived in this way, the water of the human body is a circulating system in perpetual renewal, passing from the intracellular system to the interstitial system, to the vascular system, and vice versa, constantly, but in such a way that what leaves one system is immediately replaced by a similar quantity that passes in the opposite direction from the other system to the first, both in volume and in chemical composition. When there is an alteration such that this exchange is disrupted in the sense that there is a greater amount of water retained in the subcutaneous cellular tissue, edema will appear.

The water in the human body, as we have established, constitutes, in an adult of normal weight (there are variations in children and depending on the person's state of obesity), 60 to 70% of their body weight. That 60 or 70% is distributed among:

intracellular water	45-50
interstitial water	10-15
intravascular water	more or less

The most important factors that maintain the balance between the different water compartments of the body are (fig. 26.1):

1. Permeability of the capillary membrane, a permeable membrane that allows water and crystalloid substances to pass through, but only allows the passage of 5% of plasma proteins, retaining 95% of them.
2. Effective hydrostatic pressure, 28 mm Hg, at the level of the arterial segment of the capillary, which determines at that level a greater passage of water to the interstitial space, but whose pressure decreases in the intermediate capillary and in the venous segment of the capillary to have values of 20 and 12 mm Hg, respectively, which will determine at that level a reabsorption of fluid from the interstitial space to the intravascular space due to the gradient established with oncotic pressure.
3. Effective oncotic pressure, that is, 24 mm Hg, at the level of the arterial portion of the capillary, but which surely increases at the level of the intermediate portion and the venous portion (due to water having left the intravascular compartment at the level of the arterial capillary), which determines

   At the level of these portions of the capillary, a return or passage of water from the intercellular to the intravascular space will be undermined.
4. Mechanical resistance of tissues to distension by extravasated fluid; It is approximately 4 mm Hg, and will be lower the more lax the tissue is, which explains why edema begins in the more lax tissues.
5. Tissue hydrophilia, the most difficult to evaluate due to the complexity of the factors involved; It depends mainly on the concentration of sodium in the tissues, which, in turn, depends on the greater or lesser secretion of aldosterone by the adrenal glands; Likewise, the acid-base balance, the thyroid hormone and, due to its role in the regulation of water metabolism, the antidiuretic hormone of the hypothalamopituitary system intervene in tissue hydrophilicity.
6. Lymphatic return; It is responsible for the transport and circulation of the interstitial fluid.
7. Secondary aldosteronism; In all cases of edematous retention, whatever its cause, there is an increase in the secretion of aldosterone with subsequent elimination in the urine, which causes secondary aldosteronism.

   In certain pathological states such as heart failure, nephrotic syndrome and liver cirrhosis, an increase in aldosterone with greater excretion of it in the urine has been demonstrated.

   Aldosterone is an adrenal corticosteroid hormone that is involved in sodium and water metabolism and

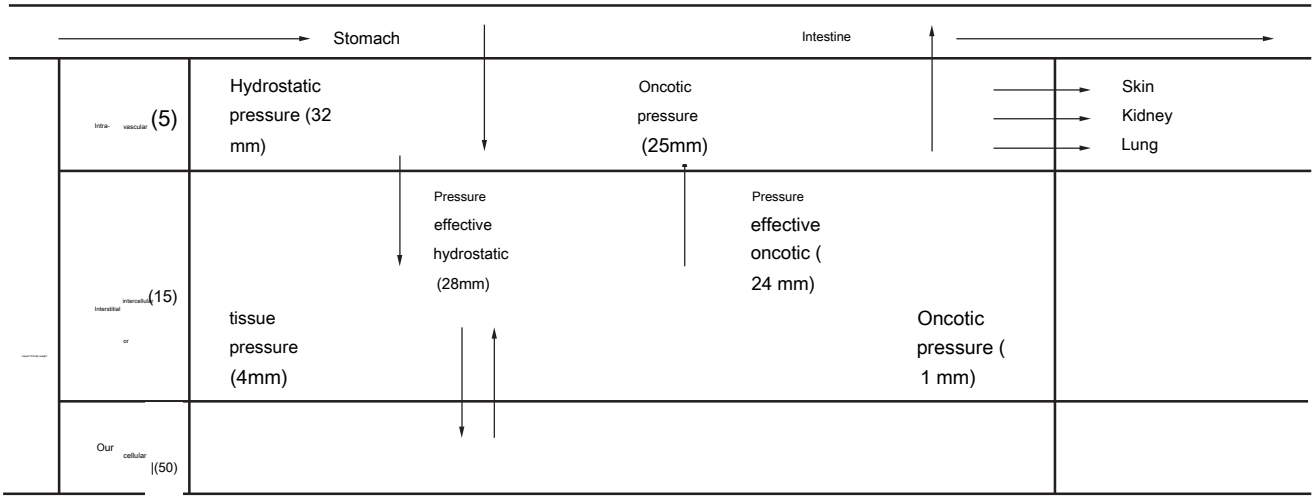


Fig. 26.1 Factors involved in water balance.

whose secretion by the adrenal cortex seems to be independent of the stimulation of the adrenocorticotropic hormone of the anterior pituitary or depend to small degrees on it, there being other extrapituitary factors, such as fluid and electrolyte changes, and especially volume, on which almost all of its secretion by the adrenal cortex depends. Let's look at these factors below.

beef:

a) Reduction in the volume of vascular fluid (potential hypovolemia) that would be present in any type of edema, by transferring fluid from the vascular to the extravascular compartment.

b) Hyponatremia and hyperkalemia, which would act as a stimulus for the release of aldosterone, whose action would determine sodium and water retention and greater potassium excretion.

SEMIOGENESIS OF DIFFERENT TYPES OF EDEMA Cardiac edema

We are not going to go into deep considerations here about the different theories that try to postulate the mechanisms of heart failure: that of Backward Failure or retrograde failure and that of Forward Failure or antegrade failure.

Due to the hypothesis of retrograde failure, there would initially be insufficient emptying of the heart with consequent retrograde stasis or behind it. The events would happen as follows:

INSUFFICIENT EMPTYING OF THE CORA

RETR GRADE STASIS (IPERVOLEMIA)

INCREASE IN CAPILLARY VENOUS PRESSURE

(WITH THE CONSEQUENT INCREASE IN IDROST TIC PRESSURE)

INCREASED TRANSUDATION

EDEMA

Due to the hypothesis of anterograde insufficiency, there would initially be a decreased systolic volume with the following sequence of events:

DECREASED SYSTEM VOLUME

DECREASE IN RENAL FLOW (OLIGURIA WITH THE CONSEQUENT RETENTION OF SODIUM WATER)

IPERVOLEMIA

INCREASED CAPILLARY VENOUS PRESSURE

INCREASED TRANSUDATION

EDEMA

In reality, this distinction is meaningless because in a closed circuit it is inevitable that the inability of the heart to maintain stroke volume (anterograde insufficiency) and the accumulation of blood in the venous portion (retrograde insufficiency) evolve together. Therefore, these two mechanisms certainly do not act independently, but, on the contrary, complement each other to give rise to initial training.

of cardiac edema; They are joined by other factors such as

As:

-Increased capillary permeability due to anoxemia of the capillary walls.

Secondary aldosteronism previously explained.

The facts can be related schematically in the following way:

Cardiac edenma begins as a consequence of the increase in hydrostatic pressure at the venous end of the capillary, which causes water to escape into the extracellular compartment, reducing the circulating volume.
This reduction in volume decreases, in turn, renal blood flow, which stimulates the release of renin by the juxtaglomerular apparatus. Renin acts on plasma hypertensinogen, forming angiotensin I, which is transformed into angiotensin II by the converting enzyme. Angiotensin II, a vasopressor, stimulates the secretion of aldosterone, which acts at the level of the distal convoluted tubule, and increases the reabsorption of sodium and water. This sodium retention creates extracellular hypertonia that stimulates the hypothalamic osmoreceptors, which determine the secretion of antidiuretic hormone and this increases renal water retention. The water and sodium thus retained by the hemodynamic failure pass into the interstitium, perpetuating the mechanism of edema that increases in the subcutaneous tissue and serous cavities. This leads to an electrolyte imbalance, with a decrease in extracellular sodium and an increase in intracellular sodium.

Kidney edema

We will study two types, that of nephrotic syndrome, and that of acute nephritis or acute diffuse glomerulonephritis.

Nephrotic syndrome. Whatever its cause, it is determined by an increase in the permeability of the basement membrane of the glomerulus, which will allow protein to escape through the urine. This proteinuria or albuminuria will result in hypoproteinemia, with a consequent decrease in oncotic pressure determined by plasma proteins and the outflow of intravascular fluid into the interstitial tissue. Furthermore, here there would also be, as mentioned above, secondary aldosteronism, probably due to hypovolemia and potential circulatory insufficiency.

Acute nephritis or acute diffuse glomerulonephritis. Its mechanism is a little more complex and not well elucidated; there seems to be a universal immunoallergic capillaropathy with the consequent increase in capillary permeability. Furthermore, spasm of the afferent arteriole and obstruction of the capillary network of the glomerulus decrease filtration. glomerular cell, which, upon passing into the tubule, undergoes the reabsorption of water and sodium. The increase in sodium concentration in the extracellular fluid would stimulate the release of antidiuretic hormone with subsequent water retention.

Edema of epatic cirrhosis

Its production mechanism is complex. First, there is hypoproteinemia due to deficient serine production in the liver with consequent reduced oncotic pressure. Furthermore, the coexistence of portal hypertension causes ascites (rich in albumin) to occur due to increased capillary permeability and loss of lymph through Glisson's capsule. Finally, insufficient catabolization of antidiuretic hormone and aldosterone due to the liver being injured contribute more to fluid retention.

Nutritional edema

Inadequate ingestion of albumin will produce hypoproteinemia and a drop in oncotic blood pressure, leading to the leakage of intravascular fluid into the interstitium, which causes edema. In vitamin B deficiencies, hypoproteinemia is associated with myocardial disorders caused by the deficiency of said vitamin which, as we know, is part of the coferments necessary for energy production, also damaging nerve fibers.

Localized edema

It is mainly due to the decrease in lymphatic circulation and the increase in venous pressure in the affected segment, either due to obstruction or blockage of the lymph nodes, in the first case; or due to obstruction or compression of one or more venous trunks corresponding to a segment of the body. These edemas that occur due to mainly mechanical circumstances are called mechanical edemas.

Localized edema may also be due to an increase in capillary permeability limited to a single area or region, rather circumscribed, due to an inflammatory (inflammatory edema) or allergic (angioneurotic edema) cause.

In summary, edema localized in a given area suggests circulatory, lymphatic or venous obstruction and/or capillary damage of an inflammatory or allergic nature. The presence of generalized edema requires continued clinical investigation to look for evidence of disease: cardiac, renal, hepatic or nutritional deficiency (primitive or secondary).

That the increase in intracapillary hydrostatic pressure and the decrease in the oncotic tension of plasma proteins (primary factors in the production of edema) are capable of determining the passage of fluid from the

intravascular partitioning to the interstitial, which is also called intercellular, is evident and beyond any discussion. On the other hand, the mechanism by which they are triggered and act, and the exact way in which they do so: the decrease in glomerular filtration; The increase in the secretion of neurohypophysial antidiuretic hormone and aldosterone from the adrenal cortex, which determine greater reabsorption of water and salt by the tubuli enal, increasing tissue hydrophilicity and water retention, include a variety of hemodynamic and hormonal mechanisms whose nature, although clarified by recent research, cannot be explained by any isolated hypothesis.

SEMIOTECHNICS AND SEMIODIAGNOSTICS

It is important to know that for edema to be visible, generalized fluid retention throughout the body (overt edema) must be 10% of body weight (edema threshold); Below this limit, fluid retention goes unnoticed (occult edema or preedema) and can only be evident by weighing the subject periodically. In this sense, daily weight variations between 50 and 100 g in an adult should not be valued, since they may correspond to normal weight variations; but sudden variations of 200 g or more per day are an index of water retention, if the variation is due to increase; or elimination of retained fluid if the variation is due to a decrease, and is accompanied by an increase in diuresis.

Once the edema becomes noticeable, its positive diagnosis will be made by verifying subcutaneous fluid retention through physical examination.

Upon inspection (inspection signs of edema) of the edematous region observe:

- Morphological alterations of the edematous region.
- Edema skin color.
- Special appearance of the skin of the edematous region.

Morphological alterations of the edematous region. Observe the traces or marks left on the skin by the wrinkles of dresses, garters, shoes, pillows or folds of sheets, visible on the body, extremities and face. Also see the deformity and increase in volume of the edematous region, which erases the anatomical inequalities: bony elevations and projections and depressions between them; In this regard, Bisbé considers that edema behaves like a great leveler. The legs, in cases of edema of great intensity and duration, come to acquire an appearance similar to that of elephants, which is why these edemas have been called elephantiatic edema.

Edematous skin color. The color will be red in cases where the edema is due to marked active vasodilation, as occurs in inflammatory edema or allergic, and they are called ruddy edema; blue or violet in those cases in which there is also cyanosis, as seen in the edema of heart failure, called cyanotic edema; white, when there is capillary vasoconstriction in the edematous site and the liquid accumulated in the subcutaneous cellular tissue compresses the capillary network of the dermis and displaces blood from them, which occurs, above all, in renal edema and in edema due to hypoproteinemia (renal or nutritional), calling them white edema.

Finally, there is the tan color that is observed when an increase in the skin pigment (melanin) has occurred in the skin due to the continued irritation that the edema produces due to its long duration, which is consequently seen in long-lasting chronic edema and these edema are called tan.

Special appearance of the skin of the edematous region. In recent edema the skin is thin, smooth, tense and shiny. In chronic cases it is thick, rough like a shell, sometimes infected, with eczema and even ulcerations. Other times you can see blisters and small fissures through which the edema fluid flows out.

When the edema disappears, the skin may show stretch marks similar to those on the belly of pregnant women (vergeturas). When the edema is of long duration, having occurred and disappeared repeatedly, when it definitively disappears it leaves the skin dry, thin, with long striations, following the orientation of the longitudinal axis of the limb and folds and long scales in the same direction, then it is called cracked or craquelé skin.

On palpation (palpation signs of edema) of the edematous region observe:

- Temperature.
- Sensitivity.
- Consistency.
- Humidity.

Temperature. Generally, the skin of the edematous region shows a decreased temperature due to the compression that the edema fluid produces on the capillaries of the subcutaneous cellular tissue, which lowers the amount of blood that they normally contain. Inflammatory edema, on the other hand, due to the greater vasodilation that occurs, is a hot edema.

Sensitivity. Edema is generally painless except when it occurs in areas of firm cellular tissue that does not distend easily. Inflammatory edema and

angioneurotic can be painful, especially the first one. Angioneurotic (allergic) is sometimes preceded or accompanied by a sensation of itching located in the same area of the edema.

Consistency. Depending on their consistency, edema can be soft or hard. The soft or hard consistency of the edema depends on the tension of the tissues in which it is found and the subcutaneous and even dermal fibrous proliferation that occurs in long-lasting edema.

Soft edema is that which sets in gradually, appearing recently at the time of the examination, for example: edema from nephrosis, nephritis, heart failure (initial or recent). Edemas are also soft during the period of resorption or in a recurrence due to previous distention of the tissues.

Hard edema is that which develops quickly, for example due to thrombophlebitis, acute or long-term heart failure, leg edema due to varicose veins, phlebitis, trophoedema and elephantiasis due to filariasis.

In addition to what was stated above, the consistency of the edema is also determined, at least in part, by its nature, that is, edemas due to hypoproteinemia are softer than those determined by an increase in capillary hydrostatic pressure.

It should be mentioned the particular consistency, similar to glaziers' putty, that edema due to hypoproteinemia and nutritional edema in general has.

Palpation also shows the dipper or godet (fig. 26.2), which is the most important and characteristic of the signs that palpation provides us with in edema.

It is the peculiar spherical depression that the finger leaves when compressing the edematous region.

Press firmly with the index finger, thumb or any other finger, for 5 s, on a bony plane such as the malleolar region, the inner aspect of the tibia or the sacral region. When you remove your finger, its imprint persists for more or less time, constituting the dipper.

Remember that intense pressure can cause pain, especially when pressing on the inner aspect of the shinbone. Always keep this in mind, since the effort to detect edema can cause iatrogenesis. Practice first with yourself, until you find a pressure that is firm enough, but not painful.

It is not possible to produce the dipper if the edema is not very intense, especially if the skin is elastic or when the edema is very chronic (elephantiaic), in which the proliferation of the subcutaneous tissue makes any depression impossible.

When the dipper or godet is missing, the existence of edema can be appreciated by pinching a skin fold from symmetrical regions of the body between the fingers and observing that the skin in the edematous area will be increased in thickness.

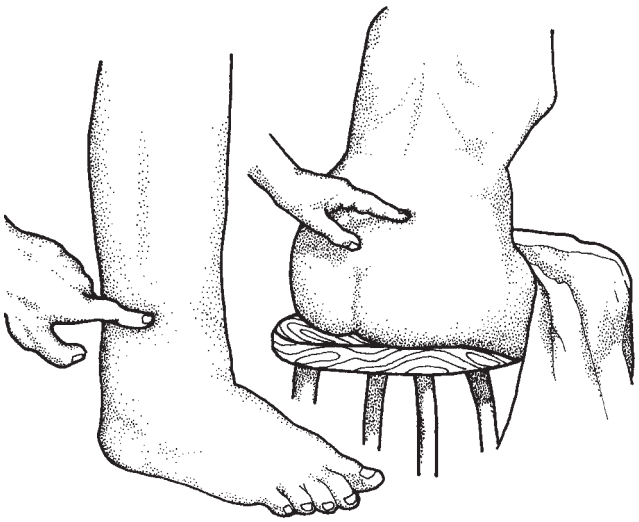


Fig. 26.2 Dipper or godet.

Humidity. Palpation, finally, informs about the degree of moisture of the skin of the edematous region; Frequently, sweating here is decreased or absent.

Place and mode of appearance, mobility, evolution, duration and manner of disappearance of the edema

Edema due to hypoproteinemia and in general all edema due to general causes, appear first in those places where the subcutaneous cellular tissue is more lax and allows itself to distend more easily and also in those places where, due to being in a more inclined position, the forces of gravity act more intensely. This is why renal edema generally appears in the eyelids, face, scrotum, foreskin, etc., but in a subject in permanent recumbency it will become more marked in the sacral and lumbar regions.

Cardiac edema, if the subject is going about his or her usual life, will begin in the feet, malleoli, and tibia.

and it will rise, as if the patient were progressively submerged in the water; but if the patient is permanently bedridden, it will appear in the gluteal, sacral and lumbar regions; or in greater intensity in those portions of the body on which the patient rests most of the time during the day.

Hepatic and nutritional edema take on a form similar to that of renal edema in their onset and distribution.

Regarding the way it appears, inflammatory-type edema always arises around an area where the classic signs of inflammation are observed, of which it is one of them: redness, heat, tumor (edema) and pain. Angioneurotic edema has a sudden onset, it acquires a more or less large volume quickly, and then disappears, generally, abruptly.

Renal edema and in general the so-called dyscratic or nutritional edema begin suddenly in the regions

of looser cellular tissue and then more or less slowly spread throughout the rest of the body, and persist for as long as the disease that produces them lasts.

Cardiac edema is slow and progressive in appearance; It begins with the most sloped portions of the body and then extends through the rest of it; It lasts as long as the cause (heart failure) that determines it. In general, it disappears slowly with treatment.

Extension of edema

Edema can be localized to a region of the body or generalized to the entire body. Likewise, they may be limited to the subcutaneous cellular tissue or true effusions may occur due to the accumulation of interstitial fluid in the cavities of the body: abdomen, pleura, pericardium, in these cases called cavitary edema.

When the edema is extended to the entire body and cavities, the patient is said to have anasarca.

When the edema is limited to the pleural cavity, the patient is said to have a hydrothorax, which may be unilateral or bilateral.

If it is limited to the pericardial cavity, the patient is said to have hydropericardium.

If the fluid collection is in the abdomen, the patient will have ascites.

When the edema is in the parenchyma of a viscus, it is called visceral edema. For example: cerebral, pulmonary, laryngeal, etc.

Localized edema. They are fundamentally due to a circulatory disorder when they are located in a limb or in a specific vascular territory. This disorder may occur due to a deficit in venous circulation caused by inflammation of its walls (thrombophlebitis);
due to compression of the venous walls themselves due to extrinsic causes (tumors), or due to the venous ducts becoming enclosed within a scarring process or adhesions that occlude them. Likewise, circulatory failure can arise in the lymphatic vessels due to inflammation of these vessels (lymphangitis); by blockage of the lymph nodes that drain a territory, whether due to inflammation (adenitis) or tumors (primitive or metastatic) or by surgery when the nodes in a region are removed.

Even more localized edema, limited to a portion or part of a segment of a limb or part of the body or to a vascular or lymphatic territory, will suggest, when accompanied by signs of inflammation, an inflammatory edema; and if it does not have these signs, in an angioneurotic or allergic edema.

Generalized edema. It is one that extends through various parts of the body, although it does not become an anasarca; example: that sits on both eyelids or on both sides of the face, scrotum, malleoli, etc. It will always make you think of a general cause: cardiac, renal, hepatic or nutritional edema. The diagnosis of each of them will be made by verifying the characteristics that we have explained before for the different varieties and by the confirmation in the patient of symptoms and signs of their main condition: cardiac, renal, hepatic or nutritional.

Registration of edema by grade

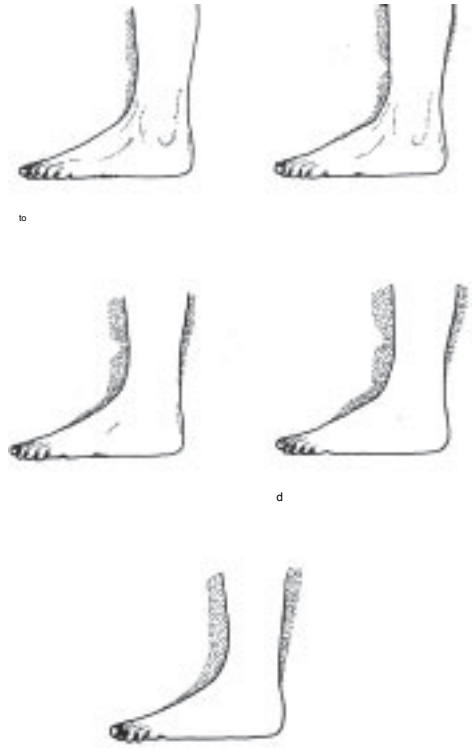
You can quantify edema in degrees or crosses (+), taking into account its semiographic characteristics upon inspection and palpation.

Edema 1 +: upon inspection all the reliefs of the structures in the area are defined. The dipper or godet is light to the touch. It is presumed that there is already a 30% increase in the volume of interstitial fluid above normal (Fig. 26.3 a).

Edema 2 +: the reliefs and contours remain more or less normal, but the cup or godet is larger and lasts longer than in the previous grade (fig. 26.3 b).

Edema 3 +: the increase in volume is obvious and the reliefs and contours of the prominences are not clearly defined. The godet is deep and is maintained for several seconds (fig. 26.3 c).

Fig. 26.3 Different degrees of edema: a, 1+;b, 2+; C, 3+; d, 4+; e, massive or intense edema.



Edema 4 +: all reliefs and contours of the area are erased. The dipper or godet is deep and is held for a longer time, possibly minutes (fig. 26.3 d).

Intense or mnastic edema: the skin surface is very shiny. As the fluid cannot move, godet is not produced and the tissues feel firm or hard (Fig. 26.3 e).

MYXEDEMA

In myxedema, the subcutaneous cellular tissue is infiltrated by a hard and elastic substance of a mucoid nature. It occurs in hypothyroidism, whether congenital (cretinism) or in the early ages of life, around the age of 40, especially in women. It is determined by a deficiency of thyroid hormones caused by atrophy of the thyroid gland or its degeneration.

It is distinguished from edema because it does not produce the characteristic dip or godet and because it is accompanied by a typical symptomatic process: dryness and peeling of the skin;
fragility and scarcity of hair, visible especially in the eyelashes or eyebrows, whose tail is generally missing (constituting the sign of the tail of the eyebrows), characteristic facies, already described; tendency to gain weight; cold intolerance, bradycardia, apathy and intellectual deficit;
very decreased basal metabolism, and very decreased radioactive iodine uptake.

In doubtful cases, myxedema should be looked for by appreciating the thickness of a skin fold taken by pinching between the fingers. The possibility of its existence should never be forgotten in order to make an early diagnosis that allows effective treatment.

SUBCUT NEO EMPHYSEMA

Here, what exists in the subcutaneous cellular tissue, determining the deformity and increase in volume of the region, is gas, almost always air.

It is identified clinically, in addition to the morphological changes that the inspection provides, by the characteristic and unforgettable crepitation that is perceived when palpating it and that is reminiscent of what is felt when compressing the lung of a corpse between the fingers.

It can be located in certain regions, or spread, becoming generalized to almost the entire body, mainly head and trunk.

It is due to infections by anaerobic, gas-producing germs (gas gangrene), in these cases it acquires very serious prognostic significance, or due to the penetration of air into the subcutaneous cellular tissue, subsequent to penetrating thoracic wounds, generally in the lung. It can also be observed in perforations of the esophagus, trachea, bronchi and lungs, where it frequently follows the spontaneous rupture in the mediastinum of the air bubbles of interstitial emphysema (mediastinal emphysema).

When subcutaneous emphysema is consecutive to mediastinal emphysema caused by any of the aforementioned causes, it acquires great intensity and is accompanied by respiratory difficulty and chest tightness, being visibly and markedly accentuated by cough when it is due to spontaneous rupture of the lung, with or without pneumothorax.

It can also be observed during some special treatments, such as in artificial pneumothorax, where it is sometimes a bothersome accident, and also in subcutaneous oxygen injections.

27

Within this chapter it is necessary to study the elevation of body temperature, which is called fever or hyperthermia; subnormal temperatures or hypothermia, and pathological variations in local temperature.

FEBRILE IPERTERMIA OR FEVER SYNDROME

CONCEPT

Fever (also called hyperthermia or hyperpyrexia) is a semiological complex in which the elevation of the central temperature of the organism above the limits accepted as physiological stands out.

Most of the time, signs and symptoms are added to the elevation of temperature, or hyperthermia, that make it constitute a syndrome, and therefore the designation of febrile syndrome is more appropriate.

SEMIOGENESIS OR FIS OPATOLOGY

Normally, man's body temperature is constant, since he belongs to the group of warm-blooded animals called homeotherms, completely different and opposite to the animals whose body temperature depends on the environment in which they are found, which are cold-blooded animals or poikilotherms.

Man's temperature is regulated by compensating the production and loss of heat, in such a way that it can be said that his temperature represents the balance between the heat produced in the tissues, or obtained from hot foods, and that emitted by the organism, which is lost in the environment. Except for the small amount of heat taken from hot foods, the production of heat in the body is mainly due to the chemical oxidation reactions that take place inside the cells during metabolism. Heat loss is caused by the intervention of physical factors, hence the distinction between chemical thermoregulation, responsible for the production of heat, and physical thermoregulation, related to the regulation of heat loss by the organism.

The mechanism of heat production, thermogenesis or chemical thermoregulation, is related to:

1. Basal metabolism, the result of oxidations in the body's cells due to cellular protoplasmic activity, which is influenced by various hormones and the nervous system.

2. The specific dynamic action of food, that is, the additional heat during digestion.
3. Accelerated visceral combustion, when an organ becomes active, as occurs in muscle and other organs, for example, the liver.

Heat loss, thermolysis or physical thermoregulation is verified by:

1. Heat irradiation, greater if the external environment is of low temperature (the physiological environment ranges between 10 and 30 °C; the optimal level is 20 °C).
2. Evaporation (insensible breathing, perspiration).
3. Thermoconduction.

The factors that increase heat production (thermogenesis) or those that increase its loss (thermolysis) intervening in caloric balance were well established by Du Bois (fig 27. I).

Controlling all chemical and physical mechanisms there are nervous centers, located in the hypothalamic region of the brain whose function is the regulation of said mechanisms, thus maintaining the almost uniform temperature, regardless of the environment, an essential characteristic of homeothermic animals.

The stimulation of the thermoregulatory centers by different factors gives rise to a higher than normal temperature, which causes fever, the causes of which can be diverse:

1. Infections. Infectious fever, the most frequent and seen in infections of all types such as: typhoid fever, malaria, septicemia, etc.
2. Surgical. Surgical fever, observed after operations of some importance carried out aseptically, and which is explained by the absorption of toxic substances released by the tissues injured in the surgical act.
3. Nervous. Neurogenic fever depends on lesions of the thermoregulatory nervous centers that give rise to dysthermia or "essential hyperthermia", with no causal relationship with what is usually understood as fever. Dysthermia is not modified by antipyretics but is modified by opiates.
4. Dehydration. Dehydration fever, observed especially in young children and due, as its name indicates, to a decrease in fluid in the body.
5. Medications. Drug fever, produced by chemical substances in general and especially some medications such as dinitrophenol, thyroxine, etc.

CLINICAL STUDY

Fever, as we have said, is a syndrome, the most important of which symptoms is hyperthermia; In addition to this, other symptoms are: dry and hot skin, as well as the mucous membranes that also dry out; chapped lips and

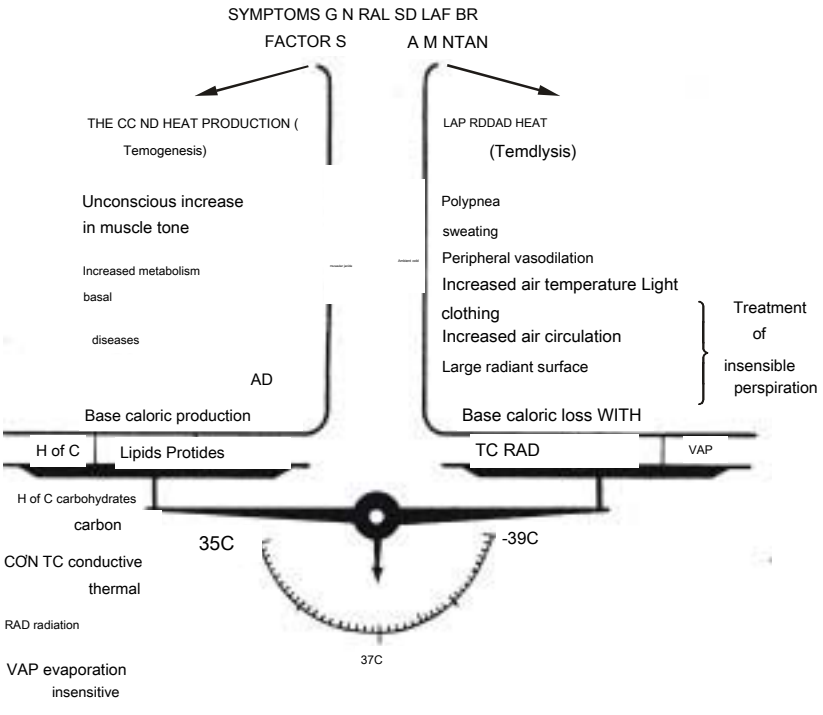


Fig. 27.1 Factors that influence caloric balance and fever (Du Bois).

bleeding, covered by scabs, called fuliginosities.

In the circulatory system the pulse rate is increased, febrile tachycardia, calculating an increase of 10-15 beats per minute for each degree Celsius that the body temperature rises, which in a certain way allows the temperature to be evaluated by the pulse. Some febrile processes break this relationship between pulse and temperature, and this is observed in typhoid fever, where there is a pulse lower than that which should correspond to the existing temperature and this is called relative bradycardia, while in other infections such as scarlet fever, a higher pulse frequency is observed than that corresponding to the temperature, which is called relative tachycardia. Variable changes in blood pressure and a state of cardiovascular erethism are also observed, with an increase in the speed of blood circulation, which explains the accentuation of heart sounds and murmurs and the presence of inorganic murmurs called febrile murmurs.

In the respiratory system, dyspnea is observed, with an increase in the number of respirations (polypnea or febrile tachypnea) and an increase in the elimination of CO, secondary to the increased metabolism of fever.

In the digestive system we find, in addition to dry lips and mouth: coated tongue, thirst, decreased appetite (anorexia), dyspeptic gastrointestinal symptoms and constipation.

In the urinary system, oliguria (decrease in the amount of urine eliminated) should be noted; The concentration and intense color of this are the most interesting symptoms with which the urinary system contributes to the febrile syndrome.

In the nervous system there are numerous symptoms, among which are: pupillary dilation (mydriasis), mental excitement with hyperideation that can lead to delirium, decay, headache and muscle and spinal pain (spinal pain) and insomnia or, on the contrary, drowsiness, which can lead to a stuporous state, characteristic of serious infections. In some adults and children it can cause seizures.

FEBRILE TYPES AND SEMIODIAGNOSIS

The daily variations of fever, that is, within 24 hours of the day, constitute the febrile types, some of them characteristic. The variations in temperature throughout the disease process constitute febrile cycles, observing in them the course of the fever or thermal curve, very characteristic in the past in many diseases, which was used for their diagnosis.

After the advent of antibiotic medication and chemotherapy these cycles are distorted, and are exceptional observation, so we will not dwell on them. The febrile types, despite the aforementioned medication, retain their validity and therefore we will describe them, at least the most important, which are:

1. Continuous or sustained fever.
2. Remitting fever.
3. Intermittent fever.
4. Recurrent fever.

We must point out from now on that the first three classify it according to the daily oscillations of the feverish period; while the fourth refers to the evolution of the fever in a given period of time.

Continuous or sustained fever. It is characterized by persistent elevations, without significant daily variations, to such an extent that the classics define it as one that has daily oscillations of less than one degree, without ever reaching the normal temperature. It is characteristic of diseases such as: bronchopneumonia, measles, dengue, influenza and certain forms of typhoid fever (fig. 27.2 a).

remittent fever. It is one in which the temperature drops one degree or more every day, but it does not return to normal either. Most fevers are remittent and this type of febrile response is not characteristic of any disease, although it is described in sepsis (Fig. 27.2 b).

Intermittent fever. It is the one that presents each feverish day, drops in temperature to or below normal. In malaria, due to its evolution in days, it can also be daily, tertiary, quartan or irregular.

If the variation between the acmé and the nadir is large enough, the daily intermittent fever is called hectic or septic fever.

Hectic fever is a needle-like fever, generally very high in the afternoon and normal or almost normal in the morning. It is accompanied by night sweats and poor general condition. It is characteristic of severe sepsis, pyogenic infections, especially abscesses, miliary tuberculosis and lymphomas (Fig. 27.2 c).

Depending on the time of day in which the remitting or intermittent fever occurs, it may be called afternoon fever, if it increases or appears in the afternoon, or inverted fever, if it increases in the morning and decreases in the afternoon. Evening fever can be found in pulmonary tuberculosis and lymphomas, among others, and inverted fever can be found in Basedow's disease or exophthalmic goiter, pulmonary tuberculosis, etc.

As can be seen, both types have been described in some forms of pulmonary tuberculosis, but they are far from being exclusive.

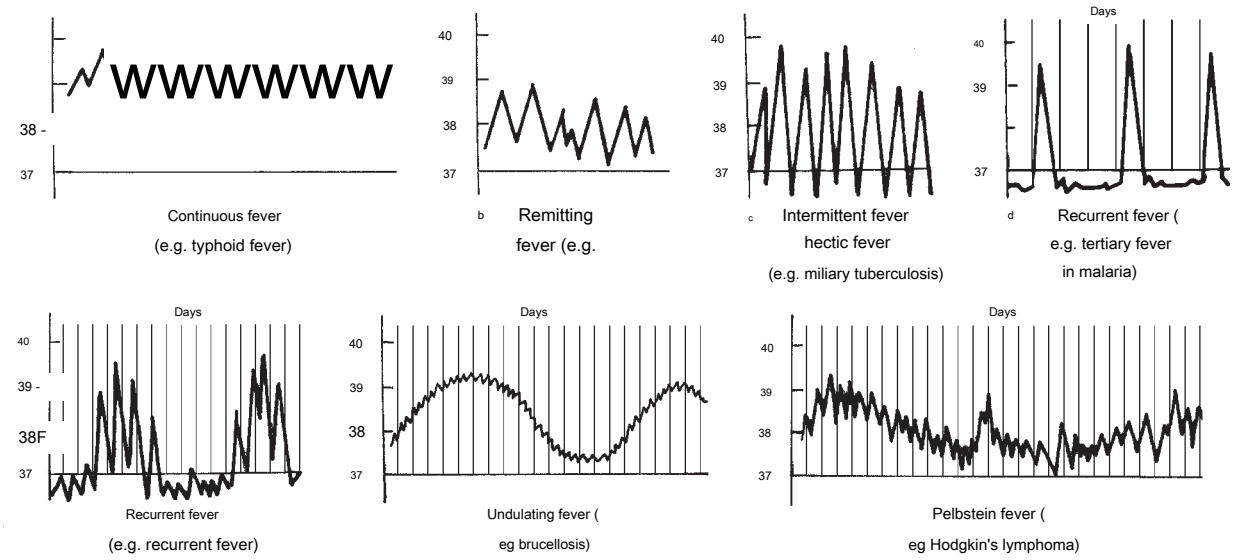


Fig. 27.2 Most frequent types and febrile cycles.

Recurrent fever. It is one in which short febrile periods occur, and at certain times, many hours of the day or up to several days, hyperthermia is absent and the patient remains with a normal temperature, although at other times he or she has a high temperature. Studied as a febrile type by many authors, in reality it is more of a febrile cycle, since it depends more on temperature variations in successive days than in the 24 hours of the day.

So a fever can be continuous, remitting or intermittent, according to the behavior of the fever during the day, and at the same time be a recurrent fever, if it evolves with cycles of febrile days and afebrile cycles.

This type of relapsing fever occurs in the following diseases:

a) Malaria: it can present in the form of tertiary fever (fig. 27. 2 d), when the fever has intervals of two afebrile days; in the form of quartan fever, when there is fever with intervals of three afebrile days, or a recurrent fever with greater irregularity, in Plasmodium falciparum.

b) Relapsing fever: disease caused by spirochetes, which does not exist indigenously in our country (fig. 27.2 e).

c) Brucellosis: the formerly called mellitense infection or Malta fever (Fig. 27.2 f), like Hodgkin's lymphoma and slow bacterial endocarditis, evolve with a type of recurrent fever called undulating fever, in which the temperature rises and falls periodically making waves.

d) In Hodgkin lymphoma: recurrent undulating fever is also called Pel-Ebstein fever and is characterized by febrile periods of 3-10 days, followed by afebrile periods of 3-10 days (Fig. 27.2 g).

e) Urinary infections with ureteral obstruction due to pus or small stones.

f) Cholangitis with lithiasis biliary obstruction: it is Charcot's " intermittent biliary fever".

g) Rat bite fever: it is due to Spirillum minus and Streptobacillus moniliforme. Recurrent fever occurs 1-10 weeks after the history of a rat bite.

h) In rare cases, localized pyogenic infections can also produce a relapsing type fever.

In general terms, the degree of the fever shows the severity of the process that determines it; Thus, an infection must be assumed to be more intense, the higher the temperature it produces. However, there are infectious processes of extraordinary severity and a high percentage of death among those who suffer from them, slow endocarditis, for example, which present with moderate fever, and there are other processes with high fever that have a benign prognosis and heal easily; example: catarrhal angina.

SUBNORMAL TEMPERATURES 0 IPOTHERMS

By this name we understand those central temperatures that offer a thermometric figure lower than normal.

SEMIOGENESIS OR PHYSIOPATHOLOGY

Depression of the thermoregulatory centers determines hypothermia.

SEMIOGRAPHY

Temperatures lower than 36.25 °C are considered subnormal. When we are called to confirm

To avoid this condition of hypothermia in a patient, the patient's temperature must be taken again, always orally or rectally, never axillary or inguinal, and only after confirming this low temperature in the mouth or rectum will we accept the existence of hypothermia, since on many occasions, the temperatures taken by the patient's relatives or neighbors are erroneous, although they cause natural alarm, requiring the doctor's presence urgently.

Temperatures that range between 35 and 36 °C are considered moderate hypothermia; intense between 33.5 and 35 °C, and extreme or deep below this last figure. When extreme prostration is added to an extreme drop in temperature, we say that the patient has a critical collapse.

SEMIODIAGNOSIS

Hypothermia is observed in: the action of very intense external cold; in critical defervescence, that is, sudden, very intense, of infectious diseases that maintained high fever, generally continuous, as in pneumonia; or in the defervescence of fever attacks in malaria. Its observation is of great importance in cardiovascular collapse, in traumatic and surgical shock, in large hemorrhages and in very cachetizing diseases such as cancer, severe anemia, etc.

LOCAL TEMPERATURE

Its study is of the greatest importance today, since it constitutes one of the means to investigate disorders. circulatory systems located in an extremity or segment thereof or in a limited part of the body. It will be discussed in more detail in the peripheral vascular system.

Local temperature is studied clinically by sensing regional temperature variations in the body with the back of the hand and, furthermore, currently by means of electrical thermometers, many of them with automatic graphic inscription. Most of the exploratory tests of these syndromes of alteration of peripheral circulation are based on local temperature variations due to different physical or chemical agents that act on the patient under investigation.

The local temperature of an extremity is decreased in the so-called peripheral circulatory insufficiency syndromes, which are produced by a decrease in blood flow to that sector of the body, whether due to functional or organic causes. Among the first we will mention Raynaud's disease, acrocyanosis, etc. Among the latter, the more or less complete obstruction of an artery in a limb due to arteriosclerosis, arterial embolism, etc.

The local temperature is increased in a limb or a region of it or a localized area of the body, whenever there is an increase in blood supply to that limb, region or area of the body, the vast majority of the time determined by capillary vasodilation. It is found in inflammation (redness, heat, tumor and pain) and in erythromelalgia, a disease characterized by capillary vasodilation at the level of the fingers.

28

ALTERATIONS IN THE REGIONAL AND SYSTEMS PHYSICAL EXAMINATIONS

REGIONAL PHYSICAL EXAM

Once the semiological study of the general physical examination is completed, we will proceed to analyze the alterations in the regional physical examination, which includes the segments or regions of the body. As most of these regions will be studied in the alterations of the different systems, in subsequent chapters, we will limit ourselves here to succinctly pointing out the most important semiological data that must be taken into account during the exploration, in the same order as it is carried out:

-Head.

-Neck.

Chest.

- Abdomen.

-Backbone.

- Extremities.

FITS

As we already know, the shape and proportions of the head vary according to constitutional type, race, age and sex; The height of the head, from the vertex to the chin, is 18-20 cm (13% of the body length), proportionally lower in men than in women and in newborns. It is also lower in Mongolians (15 9%).

Remember that the head includes the skull and the face and that the most important elements to take into account are: the position and movements of the head; the type of skull and hair, as well as the implantation of these on the forehead, in which we must take into account the shape, the grooves and the trophism; On the face, the cheeks and chin, the eyes, as well as the eyelashes and eyebrows, the nose and the ears will be explored.

When we study in detail the fascies and the endocrine and nervous systems, the most frequent semiological findings of this region will be developed.

NECK

In the neck we must explore whether there are alterations in its volume, shape, position and mobility, alterations in the vessels, such as abnormal heartbeats, noises and murmurs, and the presence of tumors.

Also, the parotid, submandibular and sublingual regions should be explored; as well as the supraclavicular region and the nape of the neck, in search of abnormalities.

In addition to the organs that run through the neck and connect the head with the trunk, other extremely important organs are found in this region: the hypopharynx, the larynx, the trachea, the thyroid and parathyroid glands and numerous ganglia.

The semiology of these organs will be described in the chapters dedicated to the respiratory, endocrine and hemolymphopoietic systems, respectively.

THORAX, ABDOMEN, VERTEBRAL COLUMN AND EXTREMITIES

Alterations in the examination of the thorax, abdomen, spine, extremities and joints will be developed in the chapters corresponding to the respiratory, digestive, osteomyoarticular and peripheral vascular systems of this Section II.

PHYSICAL EXAMINATION BY SYSTEMS

It will be developed extensively in the course of this work, which is why we refer the student to the corresponding parts in order to avoid unnecessary repetitions.

Let us only express that in each of these systems that were indicated in the clinical history we will use the different exploration methods, beginning with using the four classic methods (inspection, palpation, percussion and auscultation), either in isolation or in combination; Then we will practice the different exploratory maneuvers for the examination of the osteomyoarticular system, in its three components; of the peripheral vascular system (arteries and veins) and of the nervous system, above all, where there are multiple methods that allow studying: motility, sensitivity, tone, balance, reflexes, cranial nerves, etc. With the elements collected (symptoms and signs) we can form a syndrome and in a good number of cases establish a provisional diagnosis that can be confirmed later using instrumental methods, but which in many cases allows us to establish an immediate appropriate therapy.

Furthermore, the true doctor, the one who acquires good training in data collection and the preparation of clinical history, can carry in a simple briefcase all the essential elements to achieve this purpose, which will allow him to carry out his profession with dignity even in environments where he does not have complex equipment within his reach and carry out his mission of preventing and curing man, generically speaking, wherever he is.

That is why we have always insisted on these aspects and advised our students so that they can develop as doctors and as men, always based on those who need our knowledge and services.

29

SOMA HISTORY

Usually the osteomyoarticular system (SOMA) is studied lightly, ignoring the importance it has, since it intervenes in all the voluntary functions of the individual, that is, in the relationship life. The endocrine and central nervous systems influence it.

The questioning of a rheumatic patient is similar to that of other patients.

PERSONAL IDENTITY DATA

Sex

There are diseases that affect the osteomyoarticular system that have a preference for a certain sex. Thus we can point out that more than 95% of gout cases are in men, while it is rare in women; The reference of Hippocrates is classic, who stressed that eunuchs did not suffer from it. Systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA), even though they are diagnosed with some frequency in males, are not the most common, with the ratio estimated at 4:1 and 9:1, respectively, and according to some statistics.

On the other hand, ankylopoietic spondylitis is observed more frequently in men, although its presence is becoming more common in women every day.

Age

Age is a factor that helps us in the diagnosis. Systemic lupus erythematosus and rheumatoid arthritis most frequently affect young women of reproductive age, although in both cases they can occur at any age. Gout is almost always observed after the age of 40. Osteoarthritis, known by many as a degenerative disease, occurs more frequently after 40 and 45 years of age, depending on whether you are a woman or a man. It can be seen in younger individuals when it responds to a traumatic cause or deformities.

skin color

Skin color, ethnic group or race is of little significance. It is noted by some that gouty arthropathy is more common among whites or

Europeans, but more and more blacks and Asians are reported to have gout.

Occupation

Occupation can guide us from a diagnostic point of view; Subjects with high physical activity and joint overload have a greater predisposition to osteoarthritic changes. Lumbosciatalgia is observed more frequently in farmers. Rigarthrosis of the thumb is most commonly seen in seamstresses.

Divers suffer aseptic necrosis due to vascular occlusions caused by nitrogen bubbles.

Flat feet are common in those individuals who remain standing for many hours, especially if they are barefoot.

HISTORY OF THE CURRENT ILLNESS

Patients with the osteomyoarticular system consult primarily due to pain. Also due to rigidity, deformities, lack of muscle strength, limitation of mobility, or due to a process that the patient does not link to this system. We will analyze the most important symptoms.

Pain

Of all the symptoms that the patient tells us, pain is undoubtedly the most important.

For obvious reasons we are going to delve into some issues.

More than 90% of patients who attend Rheumatology consultations complain of pain. According to Rotés-Querol, in the Catalan region, the public "has turned pain into a synonym for rheumatism and the rheumatologist into the pain doctor."

The pain syndrome must be addressed quickly based on its etiology, its pathological substrate and the pathophysiology involved in it.

Pain prophylaxis can be treated in a primary, secondary and tertiary way. In primary school we avoid the appearance of functional or organic diseases that immediately or indirectly cause pain. Secondary prophylaxis prevents pain from becoming chronic or recurrence; The tertiary limits the impact of the pathology that causes the pain and treats it as a symptom-disease.

Semiogenesis or pathophysiology

The combination of electromechanical events involved in the perception of pain is called nociception, which groups together four pathophysiological processes that are: transduction, transmission, modulation and perception; the last two are of greater importance.

Tissue injury releases bradykinin and histamine, which stimulate cutaneous nerve endings, giving place to the appearance of pain. The intensity of the pain depends on the metabolic activity of the affected tissue; For this reason, the joint pain is severe, since the metabolism in this area is very intense.

Pain receptors (free nerve endings) are distributed not only throughout the skin, but also throughout the joint surface, the periosteum, the arteries and meninges. The rest of the deep tissues do not have many nerve endings and these are scattered; Only if the injury is extensive can it cause pain due to additive effects.

The sensory fibers penetrate through the posterior roots of the spinal cord and ascend through the lateral spinothalamic and spinotectal tracts, reaching the reticular substance, the medulla, the pons, the peduncles and the posterior nuclei of the thalamus. Some fibers ascend to the cortex.

The anatomical structures involved in the modulation are the cells of the substantia gelatinosa of Rolando; These regulate the messages that arrive from the peripheral activating fibers.

The thalanmus, the cerebral cortex and the corticomedullary projections are the main structures related to perception; This implies the function of integration of all the impulses that arrive with the elements that are the patient's own.

A delta fibers, A beta fibers and C fibers are involved in transmission, and mechanical, thermal and nociceptor receptors are involved in the transduction process.

Information processing occurs in three stages: fast, slow and prolonged, which can last days or months and is characterized by chronic changes that appear in sensitivity and somatotrophic organization. This stage occurs mainly when there are injuries to the peripheral nerves or dorsal roots.

Semiography

When talking about pain, it is necessary to know the following characteristics:

1. Intensity.
2. Location.
3. Irradiation.
4. Rhythm and schedule.
5. Periodicity.
6. Relationship with exercise and rest.
7. Relationship with environmental changes.
8. Relationship with trauma.
9. Way to calm him down.

Of these characteristics, it is important to highlight the intensity, depending on whether the pain is superficial, deep or referred.

Superficial pain is characterized by being defined, with precise limits; the patient locates it exactly: it can be exacerbated or provoked by direct stimulation of the painful area.

The structures located near the body surface can determine this type of pain, since these Areas are very rich in nerve endings. On the other hand, deep pain constitutes a dull, poorly limited, somewhat indefinite sensation located, as its name indicates, deeply in any area of the body. It generally manifests itself on deep palpation and compression.

The characteristics of bone pain are very variable, that is, they have different degrees of intensity, and may even be absent, as happens in Paget's disease, or be continuous and brutal, as happens in neoplasms. Osteomuscular pain of lancinating or compressive quality is not related to a specific entity. In many bone diseases, the pain is diffuse and lacks systematization, such as myeloma, Paget's disease, hyperparathyroidism and osteoporosis.

Referred pain is so called because it appears at a distance from the painful stimulus that caused it. It is common when there is a visceral condition with irritation of the visceral afferent pain fibers that seem to synapse in the spinal cord with the neurons that conduct cutaneous (superficial) pain, corresponding to the dermatome that embryologically establishes a relationship with said viscera, so the pain can be felt at a distance.

Sometimes, accompanying symptoms are associated with the pain characteristics described above:

a) Local or joint manifestations: heat, redness, fluctuation, crepitation, increase in volume or swelling.

b) General manifestations: fever, laxity, anorexia, weight loss.

c) Remote manifestations: acroparesthesia.

Pain of bone origin is accompanied by swelling in tumor diseases or not; The skin overlying the swelling of bone sarcomas takes on a cyanotic red color and the veins become engorged. In acute osteomyelitis, the swelling has the characteristics of acute inflammation.

Semidiagnosis

Pain of bone origin

We observe pain of bone origin in simple and pathological fractures, in osteitis deformans or Paget's disease, in primitive or secondary bone tumors, in bone tuberculosis, osteomyelitis, etc.

Pain of muscular origin

1. Physiological: after exercises.
2. Infectious: as seen in dengue and polyneuritis.
3. Parasitic: cysticercosis and trichinosis.
4. Toxic: strychnine poisoning.
5. Traumatic: rupture of a muscle.

Pain of joint origin

We observe pain of joint origin in traumatic arthritis, in acute infections such as gonorrhea when they affect the joints (gonorrhagic arthritis), in chronic infections such as syphilitic arthritis.

Pain of joint origin is also observed in metabolic diseases such as gout; degenerative such as osteoarthritis; neoplastic such as osteosarcoma;
collagens such as rheumatic fever, rheumatoid arthritis and systemic lupus erythematosus; allergic such as intermittent hydrarthrosis, and dyscratic, when it produces a true hemarthrosis (blood spill into the joint cavity) as seen in hemophilia.

Pain produced in the periarticular structures

We will observe pain of periarticular origin in bursitis, in periarticular calcinosis of hypoparathyroidism and in Stieda' s disease, which is the calcification of the internal lateral ligament of the knee.

functional impotence

Concept

Inability to perform the normal functions of a given organ.

Semiogenesis

Functional impotence occurs when the patient has a reduced ability to perform one or several movements, or there is a decrease in their strength. The cause may be of osteomyoarticular origin with integrity of the nervous system.

It must be specified which joint is the one that presents movement limitation and what type of its function is altered or diminished. When the limitation of movements is of neurogenic origin, paralysis or paresis is present.

Functional impotence occurs due to the following causes:

1. Pain.
2. Mechanical obstacle.
3. Muscle deficit.
4. Neurological disorders.

Semidiagnosis

All conditions capable of causing SOMA pain can give us functional impotence; In osteoarthritis, this symptom not only occurs due to pain, but also due to blockage of joint movement due to the presence of marginal osteophytes (growth of the bone ridge in the shape of a parrot's beak). Bone fractures are usually the most frequent causes of functional impotence.

Muscle deficit causes functional impotence in primary amyotrophies or muscle rupture.
Finally, diseases that affect the nervous system, such as acute or chronic anterior poliomyelitis and polyneuritis, also produce functional impotence.

Muscle stiffness

During the examination of cases with muscle spasm it is possible to detect the presence of muscle rigidity (stiffness), usually caused as a response to
ta to pain.

This contracture, a true tonic response of the spinal cord, tends to limit movement and causes accumulation of lactic acid, as well as ischemia due to vascular compression, which increases the condition and gives rise to a true vicious circle. In inflammatory joint processes, fluid accumulates in the periarticular tissue, especially during the hours in which the patient sleeps and since joint mobilization does not occur, which is what facilitates lymphatic drainage, immobilization results in muscle rigidity.

PERSONAL PATHOLOGICAL HISTORY

Infections of the upper respiratory tree, urethra and gynecological can sensitize the patient and lead to joint processes. Infections are known tions due to group A beta hemolytic streptococcus, as a history of rheumatic fever (RF).

Among reactive arthritis we also find an infectious history in the digestive and urogenital tracts. Yersinia, Salmonella, Shigella, Campylobacter, are the germs involved in enteric conditions that can precede reactive arthritis. However, the most common are arthritis secondary to urogenital processes, where the causative germ is Chlamydia trachomatis.

It has been shown that Chlamydia pneunoniae also classifies as a causal agent of reactive arthritis. All these bacteria produce antigens, which can be detected in the joint, by immunological techniques, producing a local immune response.

Sexually transmitted diseases (gonococcus, HIV) also cause arthritis.

FEEDING

Nutritional deficiency can cause SOMA disorders; bone pain is also common in hypervitaminosis A and D.

FAMILY PATHOLOGICAL HISTORY

There are numerous conditions of the osteomyo-articular system that occur in families, which is why it is very useful to ask the following question: do you have a blood relative who suffers from a condition similar to yours?

A history of deafness, spontaneous, painless fractures, and blue sclerae are important in the relatives of a patient in whom we suspect osteogenesis imperfecta.

The most common hereditary disease is ankylopoietic spondylitis, related to the leukocyte HLA-B27 antigen.

30

ALTERATIONS IN THE EXAMINATION OF THE OSTEOMIOARTICULAR

SOMA PHYSICAL EXAMINATION

After evaluating the history of SOMA in the previous chapter, and its examination techniques in Section I of "Introduction to the Clinic", in this chapter we will analyze the physical examination of SOMA for the diagnosis of its alterations.

GENERAL PHYSICAL EXAM

To perform the general physical examination, we will begin by evaluating the following elements:

Constitutional aspect

It can be useful in the diagnosis of some SOMA conditions; It has been noted that rheumatoid arthritis is more common in long-line patients and gout, on the other hand, in short-line patients.

Gouty people are almost always obese and can be carriers of high blood pressure and other endocrine-metabolic disorders, such as diabetes mellitus.

Attitude and facies

The attitude depends on the type of osteomyoarticular injury that the patient presents. A patient with advanced myasthenia gravis presents eyelid ptosis, the head is hyperextended because one of his hands is placed under the chin, while the elbow is supported by the other hand, in turn this hand and the entire upper limb are applied to the body to obtain support.

In patients with a fracture or dislocation of the shoulder, it is common to observe flexion of the arm next to the body (adduction) and the other hand on the shoulder.

Brand

One of the most constant signs in arthropathies is lameness. The gait examination allows us to detect some type of hip alteration and also exclude other processes that evolve with claudication of the lower extremities.

The constant lameness that prevents walking is a type of gait similar to that seen in coxitis; the foot briefly rests on the ground; It is observed in infectious sacroiliitis and in significant arthritic processes of the hip.

March "waving". The patient leans forward with each step. It is a lameness proportional to the intensity of the pain and the degree of vicious attitude, in flexion. It is observed in osteoarthritis of the hip.

Duck gait. The body swings successively from one side to the other, always on the one it is resting on at that moment. It is seen in bilateral congenital hip dislocation and progressive muscular dystrophy.

Trendelenburg march. It consists of a lateral inclination with each step and is seen, for example, in unilateral hip dislocation.

Go backwards. Required to climb stairs in ankylosis of the hip. A forward projection of the pelvis is seen when there is ankylosis, in spondyloarthritis.

In the coxavara, the pelvis on the affected side is raised with each step, allowing the foot to advance without tripping over the foot on the opposite side.

In fractures of the neck of the femur, walking is not possible. If the fracture is wedged, walking is possible, but with a limp.

One cause of lameness in children is Perthes disease.

Gait of gluteus maximus injury When placing the foot, the trunk is directed backwards.

Knee disorders lead to hip and knee flexion, avoiding heel support.

Quadriceps injury gait. The patient rests his hand on the anterior surface of the thigh every time he leans on that leg.

Bone, joint, ligamentous or static alterations of the foot also produce gait alterations.

When faced with a hip syndrome with shortening of a limb, we must consider several situations, from fractures, dislocations, tumors and infectious processes. In a patient with these characteristics, the length of the legs should be measured with a measuring tape, taking as reference the anterior superior iliac spine and the lower edge of the tibial malleolus.

PRIVATE PHYSICAL EXAM

The following sequence must be followed in the SOMA exam:

Inspection.

- Palpation.
- Arcs of joint mobility.

Percussion of the spinous processes of the vertebral bodies can reveal pathological alterations.

Auscultation can reveal the presence of arterial murmurs in vasculitis, tendon friction in scleroderma, or murmurs in the popliteal fossa in pseudothrombophlebitis syndrome.

Physical examination of the bones

Semiotechnics

Inspection. Remember that the inspection must be carried out comparing the right side with the left, although the alterations can also be bilateral and symmetrical.
What draws most attention is the presence or absence of bone deformities. We will also observe the existence of swelling or edema of the soft tissues and changes in skin color.

Palpation. Palpation should not be rough, but firm. We will check the pain caused, which can be diffuse or exquisite at a given point. We also corroborate the existence of deformities, diffuse or localized increase in volume, decrease in volume or bone depressions. When moving a limb segment where there is no joint, we may find abnormal mobility or crepitation, which is a sign of fracture.

Measurement. When performing a thorough SOMA examination, it is necessary to carefully measure the axial lines of the limbs to detect differences in limb length.

Bone

semiology

Semiogenesis

The mechanisms of production of deformities

Are:

1. Bone breakage.
2. Deficit of osteoblastic activity.
3. Deficit in calcium supply to the bone:

a) Due to absorption deficiency: celiac disease.

b) Due to vitamin D deficiency (necessary to fix calcium).

c) Due to increased urinary calcium excretion.

4. Degenerative process.
5. Inhibition or interruption of trophic stimuli from the anterior horns of the spinal cord (atrophy).

6. Periosteal reaction.
7. Alteration of bone growth centers.
8. Possible hypoxia.

Fig. 30.1 Cranial bone deformities in children.

Semidiagnosis

For a better understanding of the deformities we will classify them according to their semiogenesis:

1. Bone breakage: Colles fracture.
2. Deficit or increase in osteoblastic activity: osteoporosis and osteochondroma, respectively.
3. Deficit in calcium supply to the bone:
   a) Due to absorption deficiency: celiac disease.
   b) Due to a deficiency of vitamin D necessary for calcium fixation: avitaminosis.
   c) Due to increased urinary calcium excretion: hyperparathyroidism.
4. Degenerative process: osteosarcoma.
5. Inhibition or interruption of trophic stimuli: poliomyelitis.
6. Periosteal reaction: callus produced by poorly handled fractures.
7. Alteration of bone growth centers: achondroplasia.
8. Possible hypoxia: Volkman ischemic retraction, due to splints.

Bone deformity is frequently observed in bone diseases; For example, in Paget's disease an increase in volume of the skull occasionally occurs (Fig. 30.1) and in osteitis fibrosa, the skull also presents deformity.

Deformities of the spine are common in bone diseases. Usually it is an increase in anteroposterior volume of the dorsal spine that determines kyphosis. It is seen in osteoporosis, Paget's disease, osteomalacia.

In the limbs we can observe various deformities, from the rickets of early childhood to deformities of the femur.

One of the most frequent manifestations of bone diseases are pathological fractures. We must be fundamentally interested in those produced in osteoporosis, determined by the prolonged administration of corticosteroids. It is important to highlight fractures caused by fatigue or overload, which are observed in the neck of the second and third metatarsals and rarely, in the fourth.

Volume increase

The increase in volume of bone tumors occurs at the expense of hyperplasia of the bone and soft tissues. Non-tumor bone diseases can also cause an increase in bone volume.

which is common and characteristic of Paget's disease, hyperthyroidism, chronic osteomyelitis and Hand-Schüller-Christian disease.

Semiogenesis

"They were explained in the semiogenesis of the Deformities."

Semidiagnosis

The increase in volume occurs in:

1. Benign tumors (osteomas).
2. Malignant tumors (osteosarcomas).
3. Osteitis deformans or Paget's disease.
4. Hypertrophic pneumatic osteoarthropathy.

Shortening

Semiogenesis

The shortening of a limb is produced by:

1. Difference in bone development of symmetrical portions of congenital origin.
2. Alteration of the growth cartilage.
3. Inhibition or interruption of trophic stimuli.

Semidiagnosis

According to semiogenesis, we will classify the shortening as follows:

1. Due to difference in bone development: shortening of the ulna and radius.
2. Due to alteration of the growth cartilage:
   a) Localized: tumor, fracture or necrosis of the head of the femur.
   b) Generalized: achondroplasia.
3. Due to inhibition or cessation of trophic stimuli: poliomyelitis.

Elongation

Semiogenesis

The lengthening of a limb is produced by:

1. Difference in bone development of symmetrical portions (congenital).
2. Irritation or stimulation of growth cartilages (of hormonal origin).

Semidiagnosis

1. Of congenital origin: congenital elongation of the tibia.

2. Due to irritation or stimulation of the growth cartilages, as occurs in eosinophilic adenoma of the pituitary: gigantism in children and acromegaly in adults.

abnormal movements

In poorly treated fractures, a false joint is created that allows abnormal movements of both bone segments (pseudoarthrosis).

Crepitation

The rubbing of both bone fragments in a fracture produces crepitation.

Physical examination of the muscles

Muscular diseases are studied in the nervous system, but in rheumatology it is necessary to know elements of muscular semiology, due to the relationship of muscular alterations with the process of the musculoskeletal system, mainly the joints.

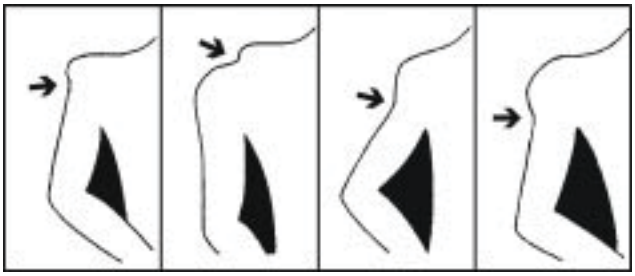
Semiotechnics

Inspection. It must be remembered that it is carried out with the patient naked, comparing both sides. We will observe the muscle volume, whether there is atrophy or localized tumors, secondary to muscle hernias or tendon ruptures, whether the muscle alteration is localized or generalized, symmetrical or not.

In the upper extremities, we will observe whether or not there is muscle atrophy or hypertrophy.

Fig. 30.2 Muscular atrophies with bone prominences

SOs.



(fig. 30.2). We will explore the interosseous muscles and the thenar and hypothenar eminences in the hands, which are frequently affected by damage to the peripheral spinal nerves.

In the lower extremities, in knee arthropathies, there is usually atrophy of the quadriceps. In addition to these muscles, the gastrocnemius and fibulas should be examined.

We will also take into account the inspection, the active movements of the muscles.

We must rule out neurological conditions such as poliomyelitis, endocrinopathies such as hyperthyroidism and metabolic diseases such as Von Gierke's disease (abnormal glycogen storage).

Palpation. We will first of all detect if there is pain on palpation, the consistency of the muscle and then we will proceed to explore passive mobility, noting if there is flaccidity or spasticity. Muscle strength against resistance will then be explored (fig. 30.3) and, finally, if deemed necessary, muscle contour measurements should be performed.

Measurement. The measurement is made to corroborate the increase or decrease in volume observed in the inspection.

Muscle semiology

They stand out:

- Increased volume.
- Congenital absence of muscle.
- Muscle atrophy.
- Caused pain.

Fig. 30.3 Exploration of muscle strength.

Volume increase

Semiogenesis

The increase in muscle volume occurs through five main mechanisms:

1. Increased muscle nutrition.
2. Increase in interstitial tissue.
3. Inflammation of muscle and supporting tissue.
4. Tumors.
5. Trauma.

Semiography and semidiagnosis

Depending on its semiogenesis, the increase in muscle volume can be:

1. Increased muscle nutrition is seen in athletes and workers engaged in rough work; It is not painful, it is generalized and symmetrical.
2. We will observe the increase in interstitial tissue in interstitial myositis. It is painful and localized to a muscle or group of muscles, with a hard consistency.
3. Inflammation of muscle and supporting tissue, as in infectious myositis. This muscle increase is painful and localized to a group of muscles.
4. Muscle increase due to benign tumors (rhabdomyoma, leiomyoma, fibromyoma) and malignant tumors (rhabdomyosarcoma, leiomyosarcoma). It is generally localized, painful and of increased consistency, according to the nature of the tumor.
5. Trauma:
   a) Due to aponeurotic rupture: muscle hernia.
   b) Due to tendon rupture: muscle retraction, as well as increase in volume; At the insertion site there is a decrease in volume.

volume decrease

Semiogenesis

A decrease in muscle volume is observed due to two main mechanisms:

1. Inhibition or interruption of the stimulus coming from the anterior horns, producing fibrous degeneration (atrophy).
2. Muscular agenesis (congenital alteration).

Semiogenesis of muscle atrophies

1. Due to alterations of the central nervous system.
2. Properly muscular.
3. Joint diseases.

Semidiagnosis

1. Atrophies caused by:
   a) Neurogenic lesions in roots and anterior spinal horns: radiculitis and poliomyelitis; in peripheral nerves: neuritis and perineuritis and in the neuromuscular motor plate: myasthenia gravis.
   b) Endocrine causes: hyperthyroidism.
   c) Muscular causes (generally corresponding to the condition of the myoneural junction): atrophic myopathies and progressive muscular
2. Agenesis: absence of the sternocleidomastoid and absence of the pectoralis major.

Muscular atrophies of nervous origin can be central and peripheral. We are interested in treating now the peripheral ones, which are of greatest interest in rheumatology.
Among them are sciatica and cervicobrachial neuralgia. Atrophies due to polyneuropathies are accompanied by pain, since they are mostly sensory-motor. They are observed in connective tissue diseases such as systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA).

Muscular atrophies, including dystrophic atrophies, are studied with other diseases of the nervous system. We will only point out myositis secondary to infections and parasitism. Also included in this group of muscle atrophies are dermatopolymyositis and interstitial myositis, although the most characteristic feature of these is fibrous contracture: in interstitial myositis, the fundamental alteration is of the connective tissue. It is manifested by moderate atrophy without a decrease in muscle strength, nor alterations in reflexes or sensitivity.

This interstitial myositis frequently accompanies:
chronic progressive polyarthritis, systemic lupus erythematosus, scleroderma and panarteritis

Caused pain

Pain of muscular origin is revealed with maneuvers or movements against resistance. For example, to check if the pain in the shoulder comes from the supraspinatus, the elbow is immobilized and the patient is ordered to execute the abduction movement; If pain appears in the shoulder we can affirm that it comes from the supraspinatus or its insertions. Similar maneuvers are valid to explore other muscle groups.

Difficulty in voluntary movements

Semiogenesis

The origin of the difficulty in carrying out voluntary movements, in a myopathy, is due to:

1. Pain.
2. Atrophy.

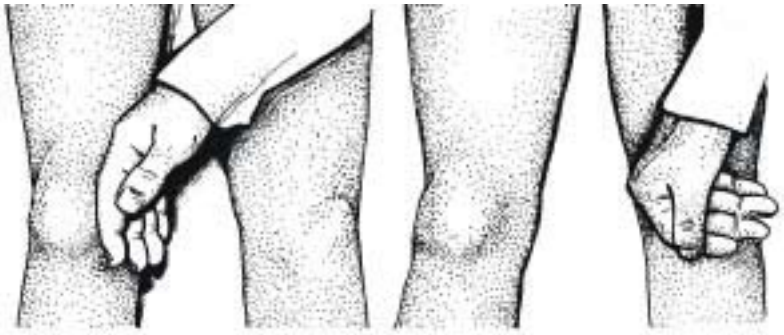


Fig. 30.4 Symmetrical palpation to detect heat.

3. Spasticity due to the increase in the intensity and frequency of the muscular stimulus on the reflex arc of tone (nervous system).
4. Flaccidity, due to interruption or interference in the reflex arc of tone.

To propose a myopathy due to atrophy, spasticity or flaccidity, it is necessary to have ruled out neurological diseases.

Semidiagnosis

After ruling out diseases of the nervous system, we will mention some diseases of the muscles that make it difficult to carry out voluntary movements.

1. Due to pain: infectious myositis.
2. Due to atrophy: myotonia.
3. Due to spasticity: myositis due to Trichinella spirallis (trichinosis)
4. Due to sagging: myasthenia gravis.

General physical examination of the joints

As we studied in Section I, the joint physical examination must be carried out with a certain systematization, which can be: the upper joints, those of the lower limbs, and the spinal column, which can be explored at the beginning or at the end. Later, after explaining the examination of the spinal column, we will deal with the semiological study of each joint separately.

The most important physical signs of the patient with joint disease are:

1. Alterations in joint temperature, consistency and color.
2. Increase in volume, as a consequence of periarticular edema or synovial thickening (synovitis). Also due to joint effusion or bone neoformation (osteophytes).
3. Diffuse or localized pain.
4. Limitation of movements.
5. Deformity.
6. Crepitation.
7. Muscle disorders (weakness, atrophy).
8. Subcutaneous nodules.

Alterations in joint color, consistency and temperature (fig. 30.4) allow us to fundamentally differentiate inflammatory processes (arthritis) from degenerative processes (osteoarthritis). Synovitis is synonymous with arthritis and is defined as inflammation of the synovial membrane.

The increase in joint volume is easily detected by inspection and verified by palpation.
It originates from an effusion, but is also caused by periarticular inflammation of soft tissues, thickening of the synovial membrane, bony protuberances or fat pads. Normally the joint contains a small amount of synovial fluid that is not detected on palpation; When it is evident, it is a sign of synovitis.
Joint effusion, as well as thickening of the synovial membrane, may be present in rheumatoid arthritis; but also in osteoarthritis, when there are osteophytes that irritate the synovial membrane, pinching it and causing synovial fluid to leak.

Also, by palpation, it is defined whether the pain is intra- or extra-articular (ligaments, bursae).

Limitation of movement is common in rheumatic processes. We have, therefore, to know the normal ranges of movement in each joint, to know if there is a limitation or not. The movements can be active and passive; Assets should be explored first.

Joint limitation can also be classified as transient, almost always due to joint effusion, periarticular stiffness and blockage due to loose bodies. The limitation can be permanent, when it is secondary to fibrous or bony ankylosis, destruction of articular surfaces or subluxations.

Joint deformity is caused by poor joint alignment; It is a consequence of the destruction of ligaments, contracture of soft tissues, joint erosions or subluxations. The lateral or external deviation is the

valgus deformity, common in inflammatory and degenerative processes. The internal or medial deviation is called varus.

Determining muscle strength can be difficult. Several systems have been described, the six-grade system is frequently used:

Grade 5 (normal) ... 100 strength full range of motion against gravity and resistance Grade 4 (good) ... 75 strength full range of motion against gravity and with moderate resistance

Grade 3 (regular) ... 50 strength full range of motion, but only against gravity

Grade 2 (poor)... 25 strength full range of motion against gravity eliminated Grade 1 (poor)... 10 strength only visible or palpable muscle contraction exists

Grade 0... There is no force or muscle contraction

Crepitation is characterized by palpable crackling sounds. It is triggered by active exercises and exploratory maneuvers; It can be thin in chronic inflammatory processes (rheumatoid arthritis), due to the contact of one cartilaginous surface on another, eroded or invaded by granulation tissue. Coarse crepitation is observed in osteoarthritis or osteoarthritis and is secondary to irregularities of the cartilaginous surface.

The use of muscles is related to the state of the joint. Muscle and joint functions are closely related.

A joint that is swollen and limited in its movement leads to muscle atrophy (disuse atrophy).

Finally, subcutaneous nodules have diagnostic significance. They are observed in rheumatoid arthritis, mainly on the extension surface of the forearms and occipital region. Gout tophi are located in the helix of the ear, elbows, hands, Achilles heel and first ball (big toe) of the foot (fig. 30. 5);
They are yellowish white in color and excrete a whitish material; Under the microscope, monosodium urate crystals are observed. Nodular formations can be found in rheumatic fever and some vasculitis.

In addition to simple radiography, we now have ultrasonography, which offers many possibilities in the detection of periarticular soft tissue injuries.
It is also useful in demonstrating intra-articular effusions, whether synovial fluid or blood. It helps extraordinarily in the diagnosis of tendon, ligamentous and muscular ruptures. The precision achieved by computed axial tomography (CT) and magnetic resonance imaging (MRI) allows for the most complex intra- and peri-articular diagnosis.

Spine scan

Semiotechnics

The complexity of the vertebral structures, as well as the interposition of powerful muscles and ligaments, make examination of the spine difficult. However, physical exploration is carried out according to the same rules as the rest of the systems.

Inspection. Remember that it will be done with the patient in a "at attention" position: standing, with arms attached to the body, feet slightly apart, with his back to us and to the light; observing the position of the pelvis, height of the shoulder and pelvic girdles, looking for asymmetry. If the pelvis is oblique, it will be necessary to investigate whether one lower extremity is shorter than the other (shortening) or if there is a vicious position of the hips. or knees, which produces a false shortening. By raising the shorter lower limb, we will restore the horizontality of the pelvis. A scoliosis that disappears when correcting the obliquity of the pelvis depends on this:

If it does not go away, the scoliosis is secondary to the spine itself.

With the patient in profile we will observe the pathological curvatures. It was already explained in Section I that the increase in posterior convexity or the presence of a convexity where there is normally none, produces a deformity called kyphosis. There may also be an increase in the posterior concavity or appear where it does not normally exist; This is called hyperlordosis or pathological lordosis, respectively.

The semitechnics of kyphosis are important. First of all we must remember that the posterior prominence of the spinous processes is called hump. If the hump is abrupt, angular, comprising one or two spinous processes,

Fig. 30.5 Subcutaneous nodules of chronic polyarthritis on the posterior aspect of the elbow.

sas, has a different meaning than the curved, regular, harmonious hump, which includes several spinous processes. An angular kyphosis always means destructive spinal or disc injury. Angular kyphosis occurs in Pott's disease (tuberculous spondylitis), compression fractures, pathological fractures (malignant neoplasms, spinal tumors, giant hemangioma, osteoporosis, osteomalacia).

Palpation. Remember that palpation of the vertebral column is performed by applying lateral movements to the spinous processes and pressing the emergency points of the nerve roots, looking for pain (see Section I). Then the paravertebral muscles are palpated, checking the degree of spasticity they may have and finally, passive movements of the spine are performed, segment by segment.

Cervical spine

Semiotechnics

Inspection. It is performed with the patient sitting to detect rectification, hyperlordosis, kyphosis and other deformities. Muscle spasm with displacement of the head to one side (torticollis) can be seen upon simple inspection.

Palpation and movement of the neck may cause pain. Compression of the neck (vertex of the skull vertically), if painful, expresses organicity;
the opposite maneuver of vertical traction should produce relief. This sign is corroborated if there is spontaneous pain that disappears with vertical traction (see Fig. 7.4).

Dorsal column

Semiotechnics

Inspection. Kyphosis, lordosis and rectification should be required in the sagittal plane and scoliosis in the lateral plane. If there is scoliosis, sometimes dorsal and other times dorsolumbar, the height of both iliac spines must be determined, since scoliosis may be due to the shortening of a limb, which causes protrusion of the opposite hip, with a certain degree of flexion (according to the shortening) and formation of a lateral curvature of the spine; In this case, the scoliosis disappears if the patient is examined in a sitting position, as well as by correcting the shortening by placing a supplement under the foot.

When the patient performs anterior flexion of the trunk, an elevation of the scapula (winged scapula) may be accentuated or revealed, secondary to the rotation of the vertebral bodies due to scoliosis (fig. 30.6).

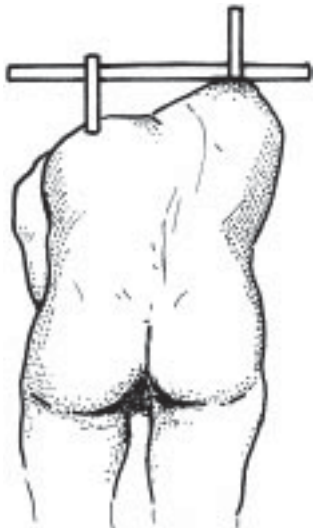


Fig. 30.6 Winged scapula.

The presence of a localized hump may be secondary to crushing, infectious spondylitis, or Pott's disease. If the hump is rounded and harmonious, it is in favor of an ankylopoietic condition or senile kyphosis.

During palpation and percussion, as in the cervical spine, mobilization of the spinous processes must be practiced and the existence of paraspinal spasms determined.

Palpation, percussion, and mobilization are combined to perform the Finck maneuver.

Finck maneuver. The patient placed in a supine position; The explorer inserts his hand under the back with the palms facing up; The spinous processes are percussed with the middle finger. If pain is caused, the maneuver is positive. It can also be performed with the patient in a prone or sitting position.

The lower limbs will be measured with a tape measure, since shortening is a cause of scoliosis.

The patient will also be asked to make lateral movements to the right and left to determine limitation in this sense.

In the dorsal region, there are practically no flexion-extension movements and the lateral ones are not very evident. Checking rib mobility in spondyloarthropathies, either by simple inspection or by measuring the chest perimeter with a tape measure, above the nipples. The increase in chest perimeter at the moment of inspiration is equal to or greater than 66 cm; In 91% of spondyloarthropathies, the figure is

lumbosacral spine

Semiotechnics

Inspection. In patients with lumbar manifestations, the weight must be specified, since the segment

lumbar is the most damaged with overweight and obesity.
We must also observe if there is scoliosis, accentuated lumbar lordosis, prominent abdomen, straightening of the spine.

When the trunk and head are flexed forward, without bending the knees, the lumbar lordosis is normally erased and a kyphosis appears; In the rectification of the lumbar spine, the rounded curve of the back does not take place.
It is of no value whether the patient touches the ground or not.

The Schober test can be useful in cases of doubt about lumbar limitation. The tip of the spinous process of the fifth lumbar vertebra is marked with a dermographic pencil, with the subject standing; At the same time we make another mark 10 cm higher, horizontally. We order the patient to do a push-up. The marks will increase the distance between each other, by several centimeters; If the separation does not reach 3 cm, we can affirm that there is a limitation to flexion.

The extension is more difficult to assess, because it is fully evident when it has not even begun.

Palpation. Palpation and mobilization of the spinous processes laterally will provide us with information about the existence or not of alterations of the vertebral body, the ligament or the interapophyseal joint.

If contracture is detected on palpation of the paralumbar muscles, this will be repeated, with the patient resting, first on one foot and then on the other. If one or both sides remain contractured, it means that there is muscle spasm, regardless of which foot the patient is resting on.

If upon inspection it is found that there is a descent of a gluteal fold, both gluteal masses are palpated to check their tone and, asking the patient to contract the buttocks strongly, they are palpated again; If flaccidity due to decreased muscle tone persists on one side, palpation is positive for nerve root involvement.

Special maneuvers:

Different maneuvers can be performed that will guide us if the patient has any lumbar condition. In them, the most important thing is not the name of the person who made them for the first time, but rather interpreting what they mean. All of them cause pain and demonstrate injuries to this segment.

a) Déjerine maneuver: positive when it produces low back pain by asking the patient to cough, which increases intra-abdominal pressure.
b) Nafziger-Jones maneuver: performed by compressing both jugulars at the same time; it is also positive, if it triggers lumbar pain, due to increased pressure of the cerebrospinal fluid and increased compression on the structures that cause the pain.

c) Neri I: flexion of the head with the patient sitting, to detect provoked lumbar pain (fig. 30.7).
d) Neri II: if there is no pain with the previous maneuver, both legs are raised alternately, keeping the head flexed (fig. 30.8).

e) Lasègue maneuver: with the patient in a supine position, the extended leg is raised (flexion of the thigh over the pelvis). It is positive if pain appears when reaching 45° (fig. 30.9).
f) Bragard maneuver: after raising the leg to the place where the pain appears, it is lowered a little lower and dorsiflexion of the foot is practiced, with the leg extended. The maneuver is positive if the initial pain is reproduced (fig. 30. 10).

In the study of the lumbosacral region of the spine, the patellar (fourth lumbar root) and Achilles (first sacral root) reflexes must be explored, and compared with the respective reflexes on the opposite side, to detect the participation of the nerve or root in the lumbosacral pathology being studied.

The patient will also be asked to stand on the balls of his feet, which is impossible if the first sacral root is involved. Dorsiflexion of the big toe when resistance is offered, as well as standing on the calcaneus, will be impossible or difficult if there is involvement of the fifth lumbar root: Finally, superficial sensitivity will be explored, according to its segmental distribution.

Semidiagnosis of decreased vertebral mobility It is observed in:

1. Disorders of the mobile segment, of a mechanical nature (disc, ligamentous, spondyloarthritic).
   In acute low back pain, all movements can be blocked, intense contracture occurs; It differs from spondylitis due to its sudden onset and short duration.

2. Infectious processes, where the limitation of mobility is marked, manifests in all movements and the muscle contracture is very intense (it is tuberculous, brucellar, typhoid, staphyloid or streptococcal pondylitis).

3. Inflammatory rheumatic processes (ankylopoietic spondylitis, senile hyperostosis).
4. Other processes (neoplasms, myeloma, vertebral collapse due to osteoporosis).

Fig. 30.7 Neri I maneuver.

Fig. 30.8 Neri II maneuver.

Fig. 30.9 Las que maneuver.

Fig. 30.10 Bragard maneuver.

The examination of the spine is not complete if it is not accompanied by a neurological examination (voluntary motility, muscle strength and tone, sensitivity and reflexes).

Semidiagnosis of lordosis and hyperlordosis

Physiological cervical and lumbar lordosis usually appear as compensation for dorsal kyphosis. Primitive lumbar hyperlordosis, in women who have had repeated pregnancies, is accompanied by atrophic and flaccid abdominal muscles, a large, pendulous or apron-shaped belly.

The decrease in lordosis is observed in disc degeneration, disc herniation, retrolisthesis, spondylolisthesis.

Lateral curvatures of the spine or scoliosis are classified into two large groups: functional or structural and organic. The first is physiological; It can be adopted by any normal child or adult; It is corrected with voluntary movements. Organic or structural diseases are not curable; they stop progressing at 18 years of age. They are classified as idiopathic or primitive (70-80%) and secondary (paralytic or post-polio, rickets, congenital, dyschondroplasia, spondyloarthropathy, osteomalacia and diseases of the nervous system), different from poliomyelitis.

The deviation of the entire spine, which appears in patients who suffer symptoms of disc degeneration, with or without sciatica, is called antalgic attitude.

Particular study of the joints

sacroiliac joint

Semiotechnics

Inspection. Affectation of the sacroiliac artery alters gait; In infectious sacroiliitis, lameness is observed with

inability to support the foot. Chronic processes, such as spondyloarthritis, can occur without lameness of the lower limbs. It is possible to find elevation of the sacroiliac line, due to ligamentous distention, as in septic arthritis.

Upon inspection, one can discover, below the posterosuperior iliac spine, an oval, painless swelling, of great diagnostic value, the osifluent abscess, exclusive of tuberculous arthritis.

Abscesses can be found in the buttock, at the starting point of the last lumbar vertebrae, at the level of the sciatic notch, from where they can reach the posterior surface of the thigh.

Increased volume of the buttock can be observed in sarcoma of the iliac bone.

Atrophy of the buttock can occur in various processes:

1. Sacroiliitis of any etiology.
2. Spondyloarthropathy; little perceptible, because it is usually bilateral.
3. Arthritis of the hip.
4. Root sciatica of L5 and SI.

Palpation. There is only one point that has great semiological importance, because it arouses pain when pressing the joint line; It is the sacroiliac point located below the posterosuperior iliac spine, at the level of the second sacral foramen, which we already talked about in Section I. In the buttock we also find the gluteus medius point, at the exit of the sciatic notch, affected in the radicular sciatica and the ischiotrochanteric point, in hip arthropathies.

Mobility, at the ages in which it is present, is scarce, so its exploration has no value.

Special sacroiliac maneuvers:

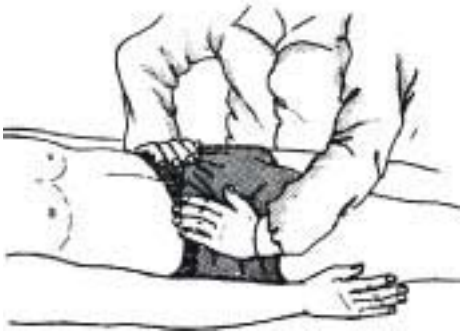
There are numerous maneuvers whose objective is to produce pain when the joint is affected. When performing them, the hips and the lumbosacral region are also mobilized, so for these maneuvers to have value, it is necessary to exclude the presence of hip conditions; This can be achieved with prior exploration of its mobility and specifying that the pain produced by the maneuvers is located in the areas of the superointernal quadrant of the buttock or in the posterior aspect of the thigh, and even in the calf. Therefore, you should ask the patient if pain occurred and what its location is.

a) Volkmann maneuver: patient in supine position;

Supported with both hands, we carry out a forced separation of both anterior iliac spines; In this way, traction is performed on the anterior sacroiliac ligament. If central pain occurs, lumbosacral processes must be ruled out (Fig. 30.11).

Fig. 30.11 Volkmann maneuver.

Fig. 30.12 Erichsen maneuver.



b) Erichsen maneuver: patient in supine position, forced approximation of both anterior superior iliac spines is performed; In this way, the posterior sacroiliac ligaments are pulled (fig. 30.12).

c) Laguerre maneuver: patient in supine position, knee and hip on the sick side flexed and abducted. The anterior superior iliac spine on the opposite side is fixed with one hand, with the other hand we rest on the flexed knee and exert pressure towards the plane of the bed.

d) FABERE maneuver (word formed by the initials in English of flexion, abduction, external rotation and evaluation): the leg of the hip to be explored is flexed and the heel of that foot is placed on the opposite knee, so that the hip is abducted. Then, with one hand we fix the opposite iliac crest and with the other we press downwards, on the internal surface of the flexed knee, bringing it against the plane of the table to cause external rotation of the hip being explored (fig. 30.13).

Patrick's sign is evident with this maneuver, when there is pain.

e) Lewin maneuver: patient in lateral decubitus, on the healthy side, we apply pressure with the hand or forearm on the iliac crest, applying the weight of the explorer's body; It is less sensitive and is positive less frequently than the previous ones (fig. 30.14).

Fig. 30.13 FABERE maneuver.

Fig. 30.14 Le in maneuver.

f) Menell maneuver: the patient is placed in lateral decubitus; the upper leg is placed in extension and the lower leg in flexion; We place one of our hands at the level of the coxofemoral joint and the other, on the rib cage; A sudden movement is then performed, forcing the iliac bone forward and the rib cage backward.

g) Maneuvers that mobilize the sacroiliac joint in shear:

- Forced hyperextension of the thigh over the pelvis. In the ventral or prone position, with the knee flexed. The sacrum is held fixed with the other hand (fig. 30.15).

Fig. 30.15 Hip extension maneuver.

Fig. 30.16 Hip flexion maneuver.

- Forced hyperflexion of the thigh over the pelvis. In the dorsal or supine position, keeping the thigh extended (fig. 30.16).

Semidiagnosis

The diagnosis of a sacroiliac syndrome needs to exclude the exploration of other regions:

1. Lumbar region.
2. Neurological examination of the last two sacral roots (superficial painful sensitivity, tendon reflexes and muscle strength).
3. Exploration of the hip.

Ankylosing spondyloarthritis is the most common cause of sacroiliitis, which may initially be unilateral, but is usually bilateral.

It is a sometimes subjective syndrome (pain in the buttock and back of the thigh). The maneuvers are rarely positive.

Tuberculous sacroiliitis, in countries where brucellosis does not exist, is the most common cause of sacroiliac involvement. There are also other processes, where sacroiliitis can be observed, such as in osteitis condensans elü, a rare disease, usually in women between 20 and 40 years old; The diagnosis is radiological.

Some cases of sacroiliac osteoarthritis have been described.

Imaging exams

Simple radiological study, with good patient preparation, is capable of helping in the diagnosis. Computed axial tomography (CAT) is an element of great value in sacroiliitis.

MRI is also an important diagnostic element. Combined studies are useful to establish the diagnosis early, which is often difficult with simple radiography.

Temporomaxillary joint (TMJ)

Semiotechnics

Inspection. Increased volume of this joint is common, but must be notable so that it can be observed upon inspection.

Palpation. Palpation is performed by placing the tip of a finger in front of the external auditory canal and asking to close and open the mouth.

Moderate limitation prevents the finger from entering the depression of the joint. The existence of pain and increased local temperature is synonymous with synovitis.

Mobility. It has three types of movements: vertical (opening and closing the mouth), anterior and posterior (protrusion and retropulsion of the jaw) and lateral movement.

The vertical is determined by measuring the distance between the teeth of the upper and lower arch, when the patient opens the mouth completely, which is normally 3-6 cm.

The lateral is measured with the mouth partially open; the jaw protrudes and moves from side to side; normal 1-2 cm.

Semidiagnosis

The joint can be compromised by mechanical problems, dental malocclusion, osteoarthritis, rheumatoid arthritis, systemic lupus erythematosus, rheumatic fever, Costen syndrome.

Cricoarytenoid joint

They are small joints, but very mobile. Inspection and mobility are done by laryngoscopy, direct or indirect. Palpation is carried out by applying pressure on the anterior part of the larynx or upper part of the thyroid cartilage.

It is involved in rheumatoid arthritis and systemic lupus erythematosus. The initial symptoms are a "feeling of discomfort" in the throat when speaking or swallowing and snoring.

Shoulder joint

Semiotechnics

Inspection. The position of the shoulder depends on the statics of the spine. Kyphotic people project their shoulders forward; In scoliotic patients there is a difference in height between both shoulders. In arthritis and acute or very painful periarthritis, the patient keeps the arm and forearm flexed and immobile next to the chest. Possible atrophies of the deltoid, supraspinatus and infraspinatus must be looked for, comparing with the healthy side.

We will observe the sternoclavicular joint looking for deformation or swelling. The muscular belly of the biceps displaced downwards is very characteristic in ruptures of the biceps tendon.

Skin characteristics may be of diagnostic use. The existence of ecchymoses on the inner surface of the arm and even the forearm are common in ruptures of the long head of the biceps. Fistulas are seen in tuberculous or other arthritis.

Palpation. The important thing about palpation is the determination of precise painful points that indicate the presence of alterations in different structures (fig. 30.17).

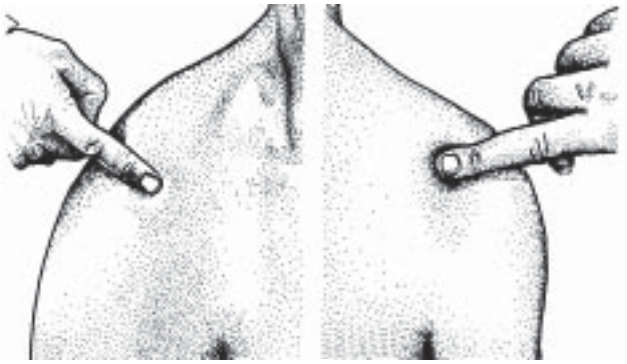
In synovial inflammatory processes it is possible to palpate painful points, in places where the capsulosynovial formation is less protected. The lower painful point, vertex of the armpit; the anterior painful point, at the level of the synovial extension that runs through the bicipital groove; a posterior point, located in the retroacromial fossa (fig. 30.18). Pain in the acromiohumeral groove, on the lateral aspect of the shoulder, is seen in subacromiodeltoid bursitis. Injuries to the rotator cuff, at the anterior end of the sulcus, may be due to involvement of the long biceps tendon.

In the adhesive capsulitis phase, no specific painful point is usually found, but there may be diffuse pain on pressure.

During palpation of the shoulder, the presence of axillary lymph nodes should be looked for when infectious arthritis is suspected.

Mobility. When we explore active mobility we must note the amplitude of the movements in degrees.
We stand behind the patient to observe the movements of the scapula. Based on the result of this exploration we can conclude the following:

Fig. 30.17 Palpation maneuvers on the shoulder.



b

Fig. 30.18 Palpation of the periarticular structure: a, anterior; b, posterior.

a) The normal amplitude allows us to exclude capsular and synovial conditions, infectious arthritis and chronic arthritis.

b) Pain during the arc of movement is an expression of injury to the rotator cuff, tendon of the long head of the biceps, subacromiodeltoid bursa.

The so-called painful arc consists of the appearance of pain when abduction reaches 80°.

c) The appearance of pain in the last 90° only is characteristic of involvement of the acromioclavicular joint.

Passive mobility is explored taking into account any limitations found during the examination of active mobility.

In infectious arthritis, chronic rheumatism and gout, a painful limitation of all movements is observed. Paradoxically, in adhesive capsulitis or "frozen shoulder" the flexion movement is preserved and pain may be absent or slight.

Subacromiodeltoid bursitis is differentiated from adhesive capsulitis, because they limit abduction around 60; On the other hand, the internal and external rotations are very little limited or free.

If mobility is normal or with little limitation, it is important to find pain in movements against resistance, with which we can discover musculotendinous injuries.

During the examination of shoulder mobility, cracking sounds accompanied by pain are of interest. They are perceived when the hand is applied to the scapula as it is dragged in the movements of the arm, and they are secondary to some irregularity in the sliding surfaces of the scapulothoracic joint.

Complementary exams

In the imaging examination of the shoulder, ultrasonography (US) can be performed, which allows us to study the soft tissues of the shoulder. We can study the bursae, the tendons and the muscles; We observed the presence of calcifications and effusions.

MRI, in combination with US, can give a complete view of shoulder injuries, allowing us to treat them effectively.

We must also mention arthroscopy, which serves as a diagnostic and therapeutic means.

elbow joint

Semiotechnics

Inspection. Remember that the elbow (fig. 30.19) normally presents a certain degree of abduction, forming an angle open outwards of about 170°, at rest. When the angle is smaller, we observe a valgus elbow and if it is greater, it is a varus elbow.

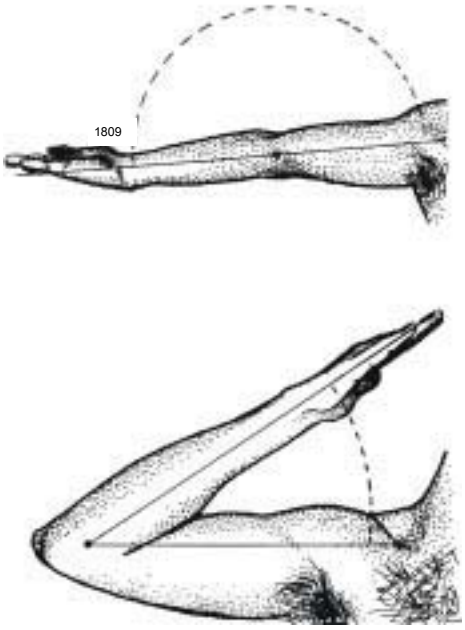
The posterior aspect of the elbow is of semiological interest, since alterations are located there that can constitute certain diagnoses. For example, rheumatoid nodules, gouty tophi, olecranon bursitis, also of gouty etiology, and psoriasis plaques. The elbow may appear swollen and red in gonococcal and tuberculous arthritis.

Palpation. The Olecranon canals provide interest from a semiological point of view. At the external end there may be pain on palpation in arthritis, mainly with effusion.

In the internal canal the ulnar nerve is palpated, when it is thickened, in polyneuritis.

Pain on palpation in the epicondyle and epitrochlea is also of great interest. In epicondylitis (tennis elbow) the characteristic painful point is located in the humeroradial interline. The diagnosis becomes more evident with maneuvers against resistance of the woman.

Fig. 30.19 Angles of normal mobility of the elbow.



wrist and hand. In epitrochleitis (golf elbow), pain occurs when the hand is flexed against resistance.

The nodes of the elbow are of great interest, as they can become infarcted in infectious arthritis of the elbow, wrist and hand.

Mobility. In this joint, the exploration of passive movements is of interest:

a) Proportionate limitation of the various directions of movement in chronic rheumatic diseases and post-traumatic arthritis. In acute processes (gout, septic arthritis) there is the same limitation, but more accentuated, reaching the suppression of movement. Mobilization is painful.

b) When there are free joint bodies, the flexion movement is limited with conservation of extension or the other way around, depending on the anterior or posterior location of the fragment. It occurs in osteochondritis dissecans, osteoarthritis, osteochondromatosis. The pronosupination movement is free.

c) A slight limitation of flexion-extension. with free rotations, it can be seen in osteoarthritis. There is no pain, but it may appear suddenly, with a sensation of bone contact. It is common in the elderly and people who have done hard work with their arms.

d) A total limitation or ankylosis occurs in chronic polyarthritis (rheumatoid arthritis), tuberculous arthritis (poorly treated), and in hemophilic arthropathy. Movements against resistance are of no interest. Flexionextension of the wrist against resistance is painful in epicondylitis and epitrochleitis.

Imaging exams

Ultrasonographic study is useful, as it can provide us with pathological images in acute processes. Simple radiography should be used in the most chronic processes and in rheumatoid arthritis, for example.

The use of arthroscopy should be limited to septic processes, fundamentally for joint lavage and drainage.

Wrist articulation

Semiotechnics

Inspection. Swelling is the first element that we can find upon inspection, which can be:

a) Located transversely, from one styloid process to the other, both on the dorsal and palmar surfaces. (rheumatoid arthritis and infectious arthritis).

b) The one that corresponds to the dorsal and palmar synovial sheaths. Its layout is longitudinal; On the dorsal side it is located above the wrist (below there is no synovial sheath); On the palmar aspect it is located above and below the wrist.

c) The swelling located laterally to the radial epiphysis corresponds to the synovial sheaths of the abductor longus and extensor pollicis brevis; It can be elongated when it corresponds to inflammatory tenosynovitis, or rounded in De Quervain's stenosing tenovaginitis.

d) The wrist is a frequent seat of the lymph nodes, a rounded tumor, resistant, not very mobile, painless, located preferably on the dorsal aspect of the wrist. In general, they are unique; They can reach the size of a chicken egg. It does not cause functional disorders, but it can cause intense pain if the cyst irritates a nerve trunk. Currently, they are considered neoplasms or cystic degenerations of the connective tissue, which undergoes central gelatinous liquefaction. They are observed in people who perform manual jobs that require precision and strength (pianists, laundresses, dressmakers). It is also common in chronic arthritis.

Palpation. A slight increase in volume can be seen, not noticeable upon inspection. Another sign that we can find is fine crepitation, when moving the fingers corresponding to the affected tendon sheaths. With some frequency, the wrist is usually affected, without any noticeable swelling, which is expressed by pain on pressure in the joint line or on movements of the wrist.

Mobility. There are two types of limitation of mobility and appearance of pain that have semiological value:

1. Proportionate painful limitation of mobility (typical of arthritis).
2. Painful limitation of only some of the movements (it is typical of the carpal ossicles or tendons).

Carpal tunnel or canal syndrome

It is the set of symptoms and signs, secondary to the affection of the median nerve as it passes through the carpal tunnel.
Signs of this syndrome include decreased sensation of the distribution area of the median nerve, Phalen and Tinel signs present, atrophy of the thenar eminence, and poor opposition of the thumb of the affected hand.

Phalen's sign: it is present if the paresthesia of the wrist worsens when it is held in prolonged flexion.

Tinel's sign: exists if palmar percussion produces stabbing or rushing pain.

When it presents with sensory and motor disorders, it is easy to diagnose. Sometimes there are only paresthesias and motor disorders are absent, so it is necessary to perform some maneuvers to be sure that we are dealing with neuritis of the median nerve (fig. 30.20).

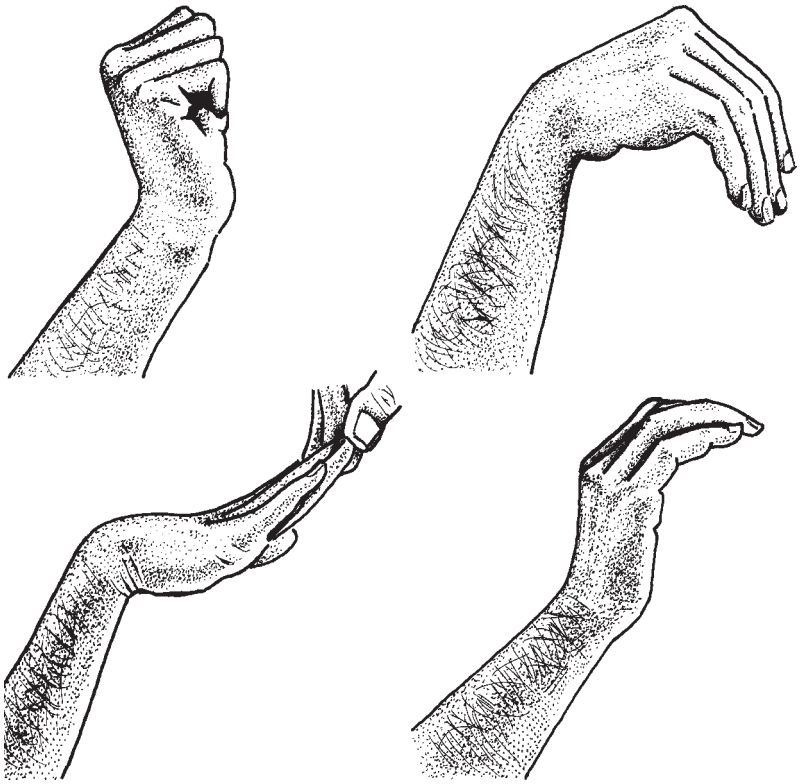


Fig. 30.20 Maneuvers for detecting neuritis of the median nerve.

Maneuvers:

a) Flexed elbow: we perform forced extension of the wrist and fingers. Next we extend the elbow. If neuritis is present, sensory symptoms of the hand appear.

b) Elbow and wrist extension: we order the patient to flex and extend the fingers repeatedly, causing paresthesias to appear.

Joints of the hand and fingers

Semiotechnics and semidiagnosis

Inspection. Upon inspection, it is observed successively:

a) Conformation of the hand: without semiological interest.

b) Skin alterations: skin disorders related to the musculoskeletal system are observed on the hand. By inspecting the skin we can also detect Osler's nodules, uratic tophi, and Heberden's nodules (fig. 30.21). Finally, Dupuytren's disease and Raynaud's disease are diagnosed upon inspection alone.

c) Swelling: it is observed only on the back, since the palmar surface, covered by the hard and rigid aponeurosis, does not allow the expansion of the swelling. This increase in volume can be diffuse or localized.
Diffuse swelling (Fig. 30.22) may be secondary to general illness; It is not accompanied by pain and does not limit mobility. If it is an expression of a local process

There is pain and limitation of movement in the affected joints (rheumatoid arthritis, infectious arthritis).

Diffuse swellings of the hand without joint involvement. They are secondary to difficulty in venous or lymphatic drainage. It is observed in breast neoplasms with resection of the axillary nodes. If we do not find an obstructing factor, then we guide the diagnosis towards angioneurotic edema, of allergic cause.

Diffuse swelling of the hand is also seen in sympathoreflex dystrophies. The positive diagnosis is made by also finding shoulder limitation and osteoporosis.
In osteoarthritis we observe localized tmefaction. They are Heberden's and Bouchard's nodules, with specific characteristics that differentiate them from other joint processes. Heberden's nodes cause enlargement of the dorsum of the distal interphalangeal joints;
They are hard, painless nodules when fully developed. Bouchard's nodules (Fig. 30.23) are also hard and painless, but are located in the proximal interphalangeal joints.

In rheumatoid arthritis, the most affected joints are the wrist, the metacarpophalangeal joints, mainly the second and third fingers, and the proximal interphalangeal joints. Furthermore, it tends to take other joints and distribute itself symmetrically.

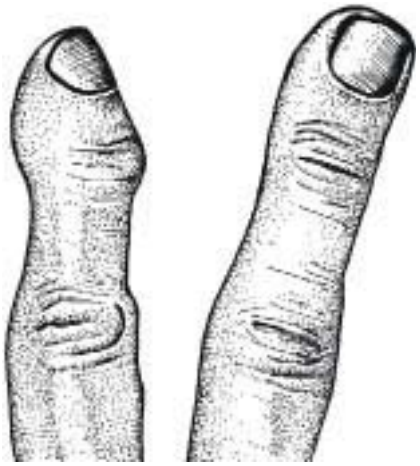


Fig. 30.21 Eberden's nodule with lateral deviation of the last phalanx.

Fig. 30.22 Joint swelling.

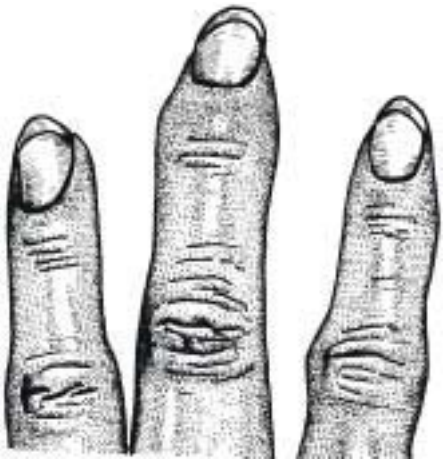
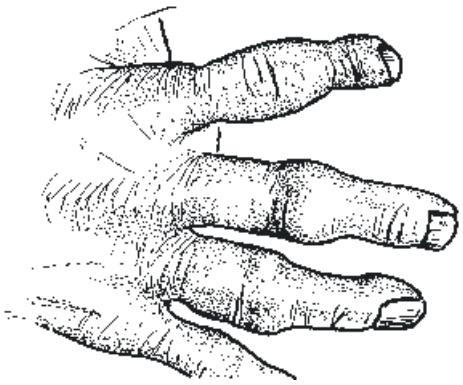


Fig. 30.23 Bouchard's nodule.



Osteoarthritis affects the distal interphalangeal joints (Heberden's nodes), and sometimes the proximal ones (Bouchard's nodes). Seizure of the trapezometacarpal joint of the thumb (rizarthrosis of the thumb) is very characteristic of osteoarthritis in seamstresses, hairdressers, typists.

The hands present special characteristics in psoriatic arthritis, gout, rheumatoid arthritis, and osteoarthritis, sometimes very difficult to differentiate clinically.

d) Diffuse or localized muscle atrophy (neurological diseases, polymyositis) (fig. 30.24).

e) Deformities are also characteristic of rheumatic hands (fig. 30. 25). The most characteristic are burst fingers, which are seen in rheumatoid arthritis and in elderly patients. In rheumatoid arthritis, "hammer finger" (permanently flexed position, due to the loss of extensor capacity), "Boutoniere deformity" and " swan's neck" finger are also observed.

In Dupuytren's disease, the deformity predominates in the last two fingers, with flexion of the first two phalanges and extension of the third.

Palpation. Palpation corroborates the characteristics of the swellings, nodules and painful points:

a) In the joints, pinching one by one between the index finger and the thumb; the metacarpophalangeals, in an anteroposterior direction and the proximal and distal interphalangeals, in a lateral direction.

b) In the palmar synovial sheaths, along their entire length.

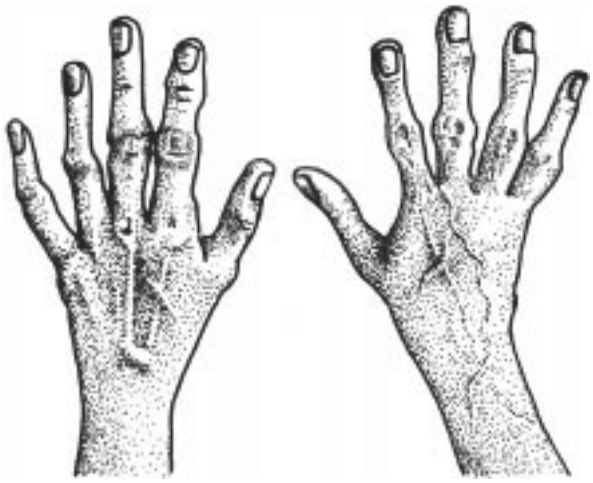
c) In the bone diaphysis.

Mobility. Mobility may be limited and painful.

When passive mobility is normal and active mobility is altered, but there is no pain, the cause is neurological or a musculotendinous tear. Limited mobility, against resistance, reveals the involvement of the muscle or tendon that we are exploring.

The abductor pollicis longus and extensor brevis are affected simultaneously at different levels of their course; pain occurs when pressing:

Fig. 30.24 Muscle atrophy.



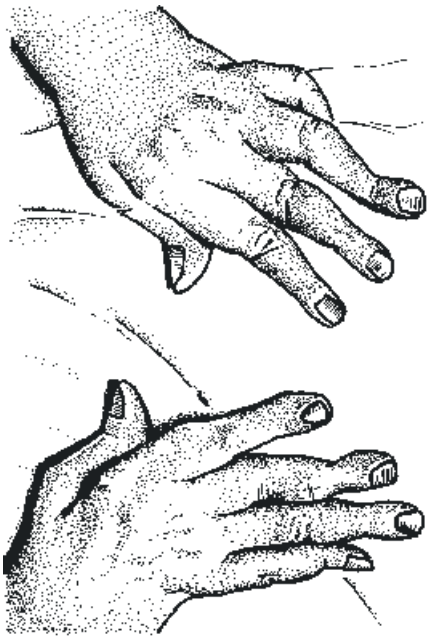


Fig. 30.25 Deformities.

a) As it passes through the canals of the lateral aspect of the radial epiphysis (De Quervain's stenosing tenovaginitis).
b) As it passes through the lateral aspect of the carpus, next to the styloid process (erroneously called styloiditis and which is really tendonitis).
c) At the insertion of the abductor longus, at the base of the first metacarpal.

Imaging exams

From the point of view of imaging diagnosis, simple radiography of the hands offers valuable diagnostic elements: periarticular osteoporosis, narrowing of the joint interline, the presence of geodes next to the cartilage, osteolytic lesions, destruction of the distal interphalangeal joints and other elements typical of rheumatoid arthritis, gout, and psoriasis. Ultrasonography and MRI can offer a first-order positive and differential diagnosis.

hip joint

Semiotechnics

Inspection. The hip is poor in local signs, because it is located deeply. By inspection it is difficult to diagnose synovial swelling, effusions, bursitis.

Septic arthritis can offer observation an increase in volume with distended skin in the area of Scarpa's triangle; Tuberculous ossifluent abscesses can also be seen at the root of the thigh, coming from the hip or pelvis. Neuropathic arthropathy can, when it produces large deformities, alter the joint configuration.

What does have semiological value upon inspection is the attitude of the thigh with respect to the trunk. This attitude should be explored with the patient in a supine position, keeping the pelvis and spine in a normal position. The anterior superior iliac spines should be in the same horizontal plane; It must be specified that the line that joins them is perpendicular to the axis of the body and that the lumbar region is in contact with the examination table or bed.

In coxarthrosis, a flexion attitude with a certain degree of external rotation and sometimes abduction in very advanced phases can be observed, radiologically demonstrable. This attitude is seen late, unlike arthritis, where it is observed early.

In coxa vara, adduction and external rotation are common and in coxa valga, abduction and internal rotation.

In hip dislocation, the attitude depends on the type of dislocation; In fractures of the neck of the femur, the attitude is in extension and external rotation.

Remember that, in addition to attitude, the inspection also explores gait.

Palpation. In the hip we must specify the location of the greater trochanter, which is important in dislocations. The search for painful pressure points is of great interest.

At the level of Scarpa's triangle, where the joint is more superficial, the painful points in the hip condition are better specified. A painful point over the trochanter is characteristic of bursitis; The movements are free except adduction.

In coxofemoral pathology, compression at the junction of the inner third with the middle third of the inguinal line causes pain.

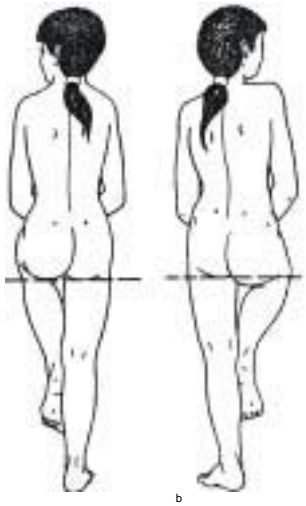
Special maneuvers:

The special maneuvers discussed in the examination of the lumbosacral spine and the sacroiliac joint can be performed in conjunction with the examination of the hip, because such evaluation is based on hip mobility maneuvers (see Figs. 30.15 and 30.16).

If you suspect a hip flexion contracture or observe restricted mobility, perform the "Thomas Test."

Ask the person to assume the supine position and bend the knee by pushing it with their hands against the chest.

The negativity of the Trendelenburg maneuver guarantees the integrity of the hip. With the patient naked, standing on his back, a line is drawn through the gluteal folds and the patient is ordered to flex one hip in the air while keeping the body resting on the other leg. If the fold of the flexed hip is below the line, the maneuver is positive for coxofemoral pathology (fig. 30.26).



b

Fig. 30.26 Trendelenburg maneuver: a, negative; b, positive.

Mobility. Active and passive mobility is explored. Active mobility was already described in Section I of this book.

Passive mobility is explored with the subject, first. in supine position. We must fix the pelvis with one hand on the anterior superior iliac spine on the opposite side and with the other hand, we move the limb that we want to explore. The movements we examine are: flexion, flexion-adduction, abduction, adduction and external rotation. The latter is performed with the knee flexed and the femur flexed over the pelvis, and causes pain when there are degenerative or inflammatory changes in the joint (fig. 30.27).

Subsequently, we place the patient in a prone position and perform the following movements: extension and rolling.

The limitations of passive mobility can be assessed as follows:

a) Limitation of all movements (infectious coxitis, rheumatoid arthritis, gout, spondyloarthritis).

Fig. 30.27 Thigh flexion and adduction maneuver.

b) In coxofemoral arthritis the limitation is late.

c) Limitation of mobility by free bodies. Variable in repeated examinations and with the characteristic of abruptly ending movement (osteochondritis dissecans, chondromatosis).

d) In coxa vara and in the protrusion of the acetabulum there is limitation in abduction.

e) Painful movements with normal amplitude (common in neoplastic metastases, osteitis of the pubis and femoral neck, trochanteric bursitis).

f) Destruction of the joint, with limitation, but without pain (Charcot arthropathy).

Complementary exams

Imaging diagnosis is essential in this joint.

The simple x-ray provides some elements. US is the obligatory complement to make a diagnosis. However, MRI is possibly the most powerful diagnostic element. Arthroscopy can be performed, without doubts of usefulness.

knee joint

The knee joint is also a support joint, so, like the hip, osteoarthritis is common there. It is one of the most complex joints, due to some of its own characteristics, among which we can highlight:

a) It is easily accessible, as it is located superficially, which facilitates its exploration, including palpation and exploratory punctures. This condition also makes it more prone to trauma.

b) The presence of the articular menisci makes it vulnerable to several conditions.

c) Its large size and its extensive joint surface and synovial membrane contribute to it being the joint that most frequently participates in inflammatory processes.

There are multiple causes and elements that can affect this joint.

Local orthopedic conditions must be managed by the clinician and the rheumatologist, to establish the different differential diagnoses.

Semiotechnics

Inspection. First of all, we observe the patient's attitude, directing attention to the knees. Upon inspection we can observe deformities: varus knees (fig. 30.28), valgus and flexion or recur- Vatumn.

Varus or valgus deformities favor the development of osteoarthritis. También es conveniente diferenciar el genu valgus, deformación de origen articular, de la deformidad ósea que se observa en la enfermedad de Paget.

La deformidad en flexión, conocida como genu recurvatum o genu flexum, es una deformidad en exten- sión que influye sobre la marchay agrava el proceso arti- cular. It is usually secondary to polyarthritis, osteoarthritis, spondyloarthritis and infectious arthritis.

En la artritis tuberculosa, además de deformidad en flexión, puede haber subluxación hacia atrás de la tibia y rotación externa (deformidad de Bonet). A slight degree of flexion may be found in Hoffa's disease.

In muscle paralysis, deformity in genu recurVatum can be observed.

By inspection we can also observe deformity due to swelling. It should always be compared to the healthy side. It is confirmed by the disappearance of eminences and depressions. These swellings can be diffuse and localized.

In diffuse swelling there is a generalized increase in volume and is almost always accompanied by synovial fluid effusion. Small spills cannot be seen by simple inspection. When there is abundant effusion, the bulge affects the entire anterior surface of the joint.

The presence of effusion is a sign of great importance and allows us to affirm that there is joint disease. It is relatively easy to verify in the knees, ankles and anterior aspect of the thorax; difficult, in shoulders and hips; and it is not perceived, in the vertebral column and in the sacroiliac joints.

It is useful to combine inspection with palpation to determine the consistency of the swelling. In the great

Fig. 30.28 Varus deformity.

In joints such as the knee, to detect minimal swelling, we must use a tape measure; A bone relief is taken as a reference point and the perimeter of the joint is measured. In large joints, differences of less than one centimeter are not taken into account.

Localized swelling has a specific diagnostic significance, being observed in:

a) Prepatellar bursitis: bulge in front of the kneecap, like a chicken egg.

b) Meniscus cyst: small, hard, resistant swelling located in the lateral joint interline.

c) Anserine bursitis: piriformis swelling, in the joint line, between the muscles of the goose foot.

d) Haffa disease: painful increase in volume of the Haffa fat pad, on each side of the patellar tendon.

e) Osgood-Schlatter apophysitis: swelling of the anterior tubercle of the tibia.

f) Tophi and nodules of chronic polyarthritis, which can be observed on the anterior surface of the knee.

Quadriceps atrophy appears in any knee condition that persists for some time.

When inspecting the posterior surface or popliteal fossa, we must take into account the longitudinal relief that normally exists when we observe the knee in extension.

It is explored with the subject in a prone position. When a localized swelling appears, we must make the differential diagnosis between a Baker's cyst, aneurysm of the popliteal artery, neuroma, adenopathy and an abscess.

Palpation. The first thing is to explore the temperature of the skin that covers the knee joint. Increased local heat can be observed in inflammatory processes, recent trauma and other processes such as hemarthrosis, infectious arthritis and gout.

In arthritis, pain can be found on pressure in the path of the joint interline and in the lateral depressions of the patella. In osteoarthritis, less frequently, there is also pain in the joint interline, but more frequently we find pain on pressure at the insertion point of the tendons of the crow's foot. In traumatic processes, the finding of painful points is more important for the diagnosis of ligamentous injuries, meniscopathies or fractures. In meniscus injury, the pain in the joint interline has the characteristic of changing position, becoming more posterior when the leg is flexed (Steimann II sign) and increases in intensity when rotating the leg towards the side being explored (Bragard sign).

Pain on pressure of the kneecap is common in almost all processes of the knee joint, such as

for example, in prepatellar bursitis. To differentiate the pain from that caused by intra-articular conditions, we must apply pressure to the kneecap, holding it by its lateral edges.

In chondromatosis, hard nodules are palpated in the peripatellar synovial fornices. Also in this location the synovium is palpated when it is thickened, a condition typical of chronic processes.

Chondromatosis nodules must be differentiated from hard nodules produced by capsular osteomas, not uncommon in osteoarthritis, which are palpated in the subquadricipital fornix.

Also through palpation we investigate the presence of effusion through three different maneuvers:

a) Maneuver I: knee fully extended. The kneecap is pressed with the middle fingers, while with the rest of the fingers of both hands pressure is exerted from above and below, as if we were expressing the liquid towards the kneecap. If the effusion is abundant, the patella can be perceived as contacting the femoral bone surface, then ascending (sign of patellar rally) (Fig. 30.29).

b) Maneuver II: the thumb is placed on the medial edge of the patella and the index and middle fingers are placed on the lateral edge; With the other hand, pressure is placed on the quadriceps tendon. If there is an effusion, some tension is transmitted to the fingers located next to the kneecap; If the effusion is abundant, the fingers are displaced. This maneuver is possibly the most sensitive.

c) Maneuver III: it is useful to discover small spills. With the palmar aspect of the fingers of one hand, press from bottom to top, at the level of the medial patellofemoral interline, with the idea of verifying emptying of the possible effusion; Immediately press from top to bottom, at the level of the lateral patellofemoral fornix; If there is a spill, the liquid moves to the other side, and a bulge is observed.

Fig. 30.29 Patella rally maneuver.

ment at the level of the patellofemoral interline, in the medial part of the joint.

Any mechanical or traumatic process capable of irritating the synovium can cause effusion, which is a characteristic, but non-specific, sign of knee conditions.

The extraction of synovial fluid and its macro and microscopic analysis is an element of great value in the diagnosis of knee conditions.

Mobility. Active mobility against resistance is useful to detect quadriceps injuries. Usually only passive mobility is explored. The subject's leg is taken with one hand and, applying the other on the knee, flexion and extension movements are created.

In flexion, the leg forms an acute angle with the thigh, less than 30°. Extension is normal when the popliteal fossa contacts the plane of the table.

During movement we can detect creaks that are better perceived with the palpating hand.

In arthritis there is variable limitation of flexion and limitation of extension results in a flexion attitude.

In osteoarthritis, the limitation is relatively less intense and mobilization is not very painful except at the end of the movement; Generally, creaks are appreciated.

In meniscus injuries, and sometimes in impacted free bodies, there is also a painful limitation of mobility, with the characteristic that they always appear in extreme positions. Blockage is characteristic of complete rupture; It is a temporary accident, with a sudden beginning and end.

Other maneuvers:

a) Mc Murray maneuver: used to detect meniscus injuries. With the person sitting or lying down, place one of your hands against the medial side of the knee to stabilize it. With your other hand, grab your ankle and rotate your leg and foot inward while trying to extend your leg. If any meniscus injury is present, the leg cannot be extended.

b) Apley maneuver or Apley Test": must be performed with the person in a prone position. It also detects meniscus injuries and foreign or floating bodies in the joint. The subject must be face down with the knee flexed at 90. Grasp the person's foot and apply pressure. Then, rotate the foot internally and externally. Locked knees or sudden, resonant sounds indicate injury or loose bodies.

c) Maneuvers to explore the ligaments: when there is a history of knee trauma, we are obliged to include the exploration of the ligaments in the physical examination.

The lateral ligaments are explored with the leg extended and performing forced adduction and abduction movements. Normally, no movement is possible. If there is a rupture or injury to the medial ligament, we will cause pain, achieving a certain degree of mobility in abduction; If the injury is in the lateral ligament, symptoms appear adduction (Fig. 30.30).

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The cruciate ligaments are explored with the leg flexed at 90°, moving the leg back and forth, as if attempting an anterior-posterior dislocation of the knee, while the foot is stabilized, with the person sitting on it1. Normally there is no movement. Pain is caused and abnormal mobility is observed when the cruciate ligaments are injured (drawer sign). Forward movement indicates injury to the anterior cruciate ligament and backward movement indicates injury to the posterior cruciate ligament.

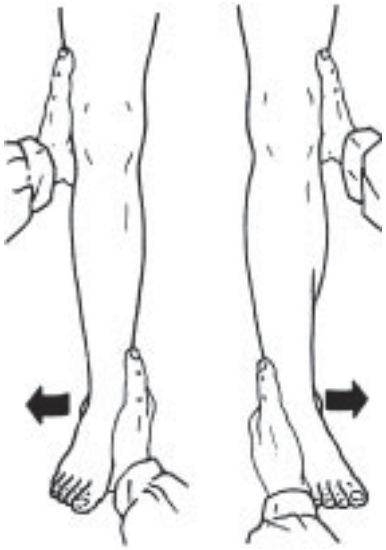
Complementary exams

Radiological studies are essential in this joint. The anteroposterior and lateral images, in the standing position, offer diagnostic elements of great importance.

In the simple x-ray, we can observe distortion of the edge of the condyles and the tibial plateau, or if there is destruction of the cartilage (osteoarthritis). Narrowing of the interarticular space is also detected, both in osteoarthritis and arthritis.

The sonographic study exposes the characteristics of the soft tissues, providing very valuable data. It reveals strokes, ligament and muscle tears.

Fig. 30.30 Examination of the lateral ligaments of the knee.



the. Tumors, cysts and loose bodies can also be diagnosed.

CT and MRI complement the diagnosis of any injury to this important load-bearing joint. Arthroscopy acquires special relevance in the knee, providing diagnostic and therapeutic elements that help resolve, in this way, just over 80% of the conditions of this joint; Allows the diagnosis and treatment of meniscus injuries and cartilage curettage.

ankle x-exploration

The ankle joint is of great interest in traumatology (fractures, dislocations and others). Rheumatic diseases also affect this joint; However, degenerative or arthritic processes are rare.

sicos.

Inspection. Conformation anomalies (varus, valgus) are associated with foot alterations. If there is a history of trauma, the presence of ecchymosis is sought. We will also look for swellings, which can be of bone, joint, ligamentous and tendon origin:

a) Bone origin: the swelling is located at the point of injury. It is generally secondary to fracture. The diagnosis is easy due to the history.
b) Articular origin: it is characterized by being located at the level of the premalleolar and retromalleolar canals, adopting an annular shape (gout, gonococcal arthritis).
c) Ligamentous origin: the swelling is located in the inframalleolar region, internal and external ligaments.
d) Tenosynovial origin: they are located at the level of the tendon sheaths located in the throat of the foot. Swelling at the level of the Achilles tendon may be due to Achilles tendinitis, bursitis, spondyloarthritis.

In intra-articular conditions of any nature, the foot is placed in a certain degree of equinus and lightness.

ro var0.

If there is pain in the calf with dorsiflexion of the ankle, it may indicate deep vein thrombosis of the leg (Homans sign).

Palpation. We will determine the existence of pain on pressure and movements, as well as heat; We will palpate the existence of swellings, which may or may not be painful or fluctuating.

In chronic synovitis we will find fungus.

In arthritis, the most painful early regions are the premalleolar canals and then the rest of the joint interline:

Mobility. It is advisable to explore active motility, which will affect the joint, muscular and tendon processes. The exploration of counter-resistance movements is also of interest.

Complementary exams

The combination of simple radiography and sonography can provide details of interest, but nuclear magnetic resonance is the most precise in the diagnosis.

Arthroscopy can be performed, although it still offers few diagnostic and therapeutic options.

physical examination of the foot

Examination of the foot is important, due to the multiple disorders caused by foot malformations. Many inflammatory rheumatisms also occur in the foot.

Inspection. With the patient standing on a hard surface, we will observe:

a) The parallelism of the feet. If the toes are directed very outwards: flat foot valgus; toes directed inward: clubfoot.

b) The height of the internal longitudinal arch. The decrease in height practically does not allow us to see the arch and the internal edge: it is observed as a straight line on the hard plane; It occurs in flat feet. Its increase is observed in the cavus foot.

c) If the heads of the metatarsals are not arranged in the form of a convex arc, dorsally. In transverse flatfoot there is a depression or depression on the dorsal aspect of the foot (dropped metatarsus).

d) With the patient on his back, we observe the Achilles heel and the posterior edge of the heel; If the foot is in a valgus position, there is an outward deviation of the heel; If the foot has a varus deformity, there is an inward deviation of the heel.

With the patient sitting we can complete the inspection and perform palpation and mobility.

Upon inspection, the foot presents a series of malformations that we can summarize as follows:

Congenital global malformations (equivarus foot).

- Acquired global malformations (flatfoot, valgus and cavus).

Malformations of the fingers (polydactyly, syndactyly, hallux valgus).

- Skin lesions (calluses, plantar ulcers).

- Localized inflammatory swelling (Köller's disease, gout).

- Other signs (Lederhane's disease, Sudeck's osteoporosis).

Palpation. Palpation in daily practice is carried out together with inspection. It is carried out by checking the characteristics of a swelling, its extension and consistency; The presence of increased temperature is checked and painful points are looked for.

The most common alterations are:

Along the longitudinal arch: flat foot.

In the metatarsophalangeal joints: chronic polyarthritis, dropped metatarsus and hallux valgus.

On the heel, at the level of the internal tuberosity: heel spurs.

On the calcaneus: osteomyelitis and apophysitis of the calcaneus, retrocalcaneal bursitis and tuberculous osteitis.

- In the pre- and retromalleolar canals: tibiotarsal arthritis.

- In the Achilles tendon: Achilles tendonitis.

Mobility. The foot can perform abduction and adduction movements, as well as pronosupination.
To perform these movements, the subtalar and midtarsal joints are required. The rest of the tarsal and metatarsal joints have little mobility.

To examine the subtalar joint, hold the leg with one hand and the heel with the other, move the heel in and out and compare it with the symmetrical joint. If movement is diminished or abolished, there is injury to the joint.

The midtarsal joint is explored by holding the heel with one hand and with the other we hold the forefoot, forming a clamp with the thumb and index finger, and we apply pronation and supination movements. The absence of these movements allows the diagnosis of tarsitis, common in gout and chronic polyarthritis.

The cancellation of the movements of the first metatarsophalangeal joint makes us suspect or verify hallux rigidus.

The foot from a pathological point of view

In addition to all the osteomyoarticular conditions that can also be found in other parts of the system, the foot, as an essential organ of support, can present alterations that are specific to it, such as:
the varus foot, the cavus foot, the calcaneal foot, the valgus foot, or combinations of these such as the equinus varus foot, the calcaneus valgus, etc. (fig. 30.31).

But more common than all these alterations is flat feet (fig. 30.32), which results from the fall of the longitudinal and transverse arches of the foot with the consequent deformation of its bone architecture.

Another frequent alteration of the foot is the so-called hallux valgus (fig. 30.33), commonly known as a bunion", which consists of a deviation of the big toe, almost always resulting from a long-standing descent of the plantar arch, sometimes as a consequence of wearing shoes that are too narrow.

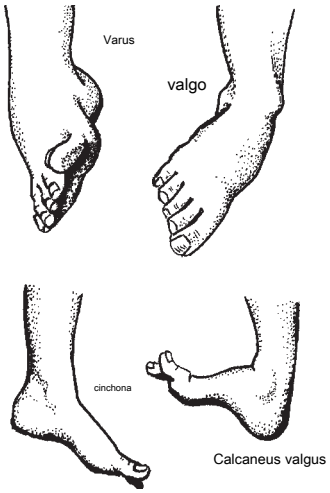


Fig. 30.31 Abnormal foot varieties.

Fig. 30.32 Foot: a, normal; b, moderate plane; C, intense plane.

Complementary exams

For foot conditions, there are special tests that help us diagnose plantar insufficiency, a frequent cause of other conditions of the foot, ankle, knees and even the hips. Among them:

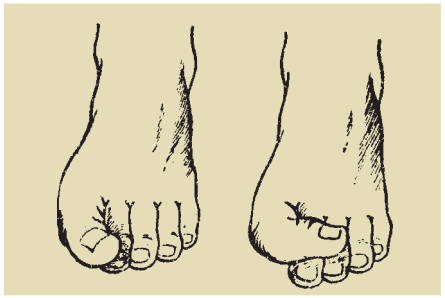


Fig. 30.33

- Podoscopy: it is a refraction system that offers us an image of the plant, with a paler color in the support areas.

Podogranma: plantar footprint.

- Photopodogram: more specialized.
- Podostatigram: more sophisticated technique.
- Radiophotopodogram.

Imaging:

Foot x-rays are not very useful, because the images overlap. Multiple studies would be necessary to locate the area we want to study. From an economic point of view, this is almost impossible, so we must combine this study with sonography to obtain some diagnostic elements.

MRI is very useful, although it is also a very expensive method and is not available to all institutions. Arthroscopy of the foot, still underdeveloped, can offer us some help. In expert hands and with their own equipment for the case, it is useful. We must not, however, abuse it.

31

OSTEOMIOARTICULAR SYSTEM. EX MENES

COMPLEMENTARY

The most important complementary tests in the osteomyoarticular system are imaging tests. Of these techniques, despite being the subject of another subject, we will point out the necessary elements for their semiological interpretation and their usefulness in the study of SOMA.

BONE RADIOLOGICAL SEMIOLOGY

The bones present an area of dense and homogeneous opacity, which is the cortex, and a spongy area with a reticular arrangement of the trabeculae.

The fundamental lesions of bone tissue from the point of view of radiological semiology are:

- Osteoporosis: increased radiological transparency and thinning of the cortical sheets.

Osteoidosis: it is a bone demineralization; The trabeculae adopt a thinner structure.

Osteoclasty: is the increase in resorption cavities in the dense bone tissue; It is accompanied by spinal fibrosis.

Osteosclerosis: is the increase in thickness of the bone trabeculae of the spongy tissue, with narrowing of the medullary cavity.

Osteonecrosis: it is the death of bone tissue; The tissue may be resorbed or sequestration may occur.

-Subperiosteal osteophytosis: it is a bony excrescence located under the periosteum.

SIMPLE RADIOGRAPHY

Before making this indication, we must carry out a complete clinical examination, which will give us guidance to know what studies we should indicate and in which part of the skeleton the lesion is located.

It is essential to take a simple front x-ray and another in profile; In peripheral joints, the general principle of examination, both clinical and radiological, must be applied: comparison with the symmetrical joint.

For economic reasons, we do not always perform the necessary number of x-rays; But what we cannot ignore is that these must be of good quality, with adequate centralization and penetration techniques.

Simple radiography with mammography technique is very useful in the study of the hands, because it allows us to observe the bone structures more clearly.

Radiological lesions can be alterations of three types: morphological, transparency and bone structure.

Morphological alterations may be due to a bone being too long or short, or very thin or exaggeratedly thick. Bends or angulations of the bone may also occur.

On the cortical surface, sometimes a radiopaque protrusion is seen, generally caused by an exostosis or benign tumor, although sometimes it may correspond to a neoplasm.

The contour of the bone may present a worm-eaten surface or more or less superficial notches, sometimes poorly defined; In these cases, it must be ruled out whether its origin is external, caused by the compression of neighboring tissues, or whether it is the result of an intraosseous, tumor, infectious or other type of lesion.

Subperiosteal osteophytosis is a common formation; it duplicates the contour of the bone, generally homogeneous, sometimes taking on a similarity to the bulb of an onion. When its opacity is such that it corresponds to the cortical area of the bone, it is called hyperostosis.

Alterations in transparency can be due to increase or decrease (the decrease in transparency is actually an increase in radiopacity).

The increase in bone transparency, in turn, can be generalized, as frequently occurs in spongy tissue, in which in some cases the trabecular pattern disappears. It predominates in the vertebrae, where demineralization causes crushing of the bone.

When the increase in transparency is localized, it generally corresponds to a tumor or infection.

If a localized radiolucency is not homogeneous, but rather uneven, it may correspond to a sequelae.

bone tro.

The decrease in transparency may be due to thickening of the bone or the appearance of a radiopaque protrusion on its surface; In general, it constitutes an expression of osteosclerosis. The entities that present these alterations are usually quite rare, with the exception of osteosclerosis.

Radiopacity is proportional to the degree of mineralization; An increase in transparency can only be seen after a loss of 25% or more of the mineral component.

Currently, densitometers are the equipment that can most accurately express bone density.

They are mainly used in the study of osteoporosis.

Alterations in the bone structure have diagnostic value only when they take a form of total disorganization, constituting the main sign of Paget's disease, in which the trabeculae are rare, very thick and are usually found in a disordered manner, without following the lines of forces of the bone.

JOINT IMAGENOLOGY

In the particular study of each joint we already assessed the usefulness of imaging and arthroscopy, so now we will refer to the diagnostic value of the imaging alterations found.

SEMIODIAGNOSIS

Although imaging is one of the pillars in the diagnosis of rheumatic diseases, it happens that in the early stages of the evolution an image that is related to the clinical picture is not usually observed.

In chronic processes, lesions appear that are very characteristic of some diseases.

Alterations in the joint interline demonstrate the existence of an inflammatory process, when it is widened. When this radiolucent space narrows and its edges become irregular, we are in the presence of a degenerative or osteoarthritis process.

Diffuse osteoporosis is seen in inflammatory arthropathies; but they can also be observed in non-inflammatory ones.

Lesions in the form of notches, bites, lacunae with an edge of osteosclerosis are sometimes observed in arthropathies such as rheumatoid arthritis and gout.

A bone excrescence in the form of a spine, spike or hook (osteophyte) is typical of osteoarthritis.

Crushing of subchondral bone tissue occurs when there is loss of resistance to pressure. It is mainly observed in the lower limbs and causes the appearance of a blurry bone contour. It is also common in degenerative processes and is also observed in inflammatory joint alterations.

Necrosis of the neighboring bone tissue promotes the appearance of bone sequestration, which provides a characteristic image, separated from the rest by a clear arc-shaped band. It is seen in aseptic necrosis and osteochondritis dissecans.

Simple x-rays are useful, in some cases alone, and in others, combined with sonography.

Contrasted radiographic studies (arthrographies) are practically out of use, because according to our experience, they are risky for the patient, sometimes because the contrast substances can irritate the synovium and on other occasions, despite the precautions taken,

Septic arthritis can occur, which can be severe.

Ultrasonography is part of imaging.
It offers us many possibilities in detecting soft tissue injuries: muscles, tendons, bursae.
It is also useful in demonstrating intra-articular effusions, whether synovial fluid or blood. It helps extraordinarily in the diagnosis of tendon, ligamentous and muscular ruptures. It is essential in the study of SOMA conditions, almost always complemented by simple x-rays.

Computed axial tomography, better known by its acronym (CAT), is a very useful study, although we should not abuse it, as sometimes it does not provide the data we need. In joints such as the sacroiliac joints, it helps a lot in verifying the damage to this joint, in our opinion, much more than special simple radiography techniques. Its greatest use is found in spinal studies.

Nuclear magnetic resonance, together with ultrasonography, are undoubtedly two important pillars in the study of osteomyoarticular conditions. Currently, nuclear magnetic resonance offers a high percentage of positivity in cases of complex diagnosis.
This imaging study is essential in herniated discs.

The precision achieved by computed axial tomography and nuclear magnetic resonance allows the most complex intra- and peri-articular diagnosis.
However, we must bear in mind that they are very expensive studies, so they cannot be used routinely. On the other hand, we cannot forget that the diagnosis of rheumatic diseases is, in the first instance, clinical and that imaging is a complement, although, in the diagnosis of rheumatic diseases in general, imaging is essential to corroborate our clinical suspicions.

Arthroscopy is another complementary investigation, which is nothing more than a joint endoscopy. It allows us In addition to reaching a diagnosis, perform therapeutic microsurgery.

EX LABORATORY MENU0

Laboratory tests must also follow a sequence that goes from:

- Hemogram with leukogram and differential.
- Erythrosedimentation.
-Calcium, phosphorus, alkaline phosphatase.
-Uric acid or urates.
- Liver enzymes.
- Total proteins and electrophoresis.
- Study of coagulation.

Even specialized imaging studies, such as:

- Antinuclear antibodies by immunofluorescence.
- Anti DNA, anti Sm, anti Ro, anti La antibodies.
- Circulating immunocomplexes.
- Lupus anticoagulant.
- Anticardiolipin antibodies.
- Rheumatoid factor.
- HLA-B27 antigen (there are many others).

We must not forget urine studies.

The study of synovial fluid is highly useful in some conditions.
Muscle biopsy is frequently used in the study of myopathies and rheumatic diseases.
Bone biopsy should be used preoperatively, especially when a neoplasia is suspected.
Skin biopsy and in some cases, kidney biopsy can also be used.
Electromyographic, echocardiographic and many more studies are sometimes necessary when studying connective tissue disease, so common in the group of rheumatic diseases.

SYNDROMES OF THE OSTEOMIOARTICULAR SYSTEM

32

From a clinical point of view, syndromes of the osteomyo-articular system can be bone, muscular, articular, connective tissue and vasculitic. Obviously we cannot delve into each one of them, but we will try to give an overview of the most important ones, so that the student is able to guide a diagnosis for a patient affected by the musculoskeletal system.

Bone syndromes almost always belong to the orthopedic area, although they frequently consult with the clinician. In this case we must continue to assist the patient, who otherwise could become disoriented and lose time in diagnosis and treatment.

As for muscular syndromes, from the field of neurologists, the same thing almost always happens as with bone syndromes.

Articular syndromes can be subdivided into articular syndromes and soft tissue syndromes (periarticular). The latter are very common in rheumatic diseases.

JOINT INFLAMMATION SYNDROME

The following entities can be grouped in this syndrome:

- Septic arthritis.
- Reactive arthritis.
- Rheumatoid polyarthritis.
- Arthropathies of secondary cause (connective pathologies)

SEPTIC ARTHRITIS

Septic arthritis is the name given to those joint conditions that are due to the presence of germs (bacteria) capable of explaining the inflammatory symptoms of a joint. It is generally monoarticular.

The condition can be caused either by direct inoculation of the joint by accident, or by a distant infection. Sometimes it occurs due to invasion of a condition neighboring the joint.

The germs that most affect the joints tend to be: staphylococcus, streptococcus, gonococcus, among others, which cause intra-articular suppuration, the most common being staphylococcus, generally following an intra-articular injection. Acute forms of gonococcal arthritis are usually polyarticular.

There are chronic forms, such as those caused by tuberculosis, in which the presence of this disease in the rest of the body is not always proven. The difficulty of detecting the bacillus in the synovial fluid sometimes requires a joint biopsy. Other conditions of this type are those caused by brucellosis, as well as viral diseases, such as rubella, mumps, mononucleosis and hepatitis.

REACTIVE ARTHRITIS

On some occasions the lesions are aseptic and these constitute the second group of the classification or reactive arthritis.

Reactive arthritis is characterized by the absence of germs in the joint; On the contrary, the persistence of an antigen constitutes the triggering factor in the pathogenesis of this infection. Arthritis occurs days or weeks after the infection, which can settle in the gastrointestinal or urogenital tract.

In the group of these inflammatory joint diseases, we can also point out rheumatic fever, secondary to a pharyngeal infection by group A beta hemolytic streptococcus. Pathogenically it is related to immunological reactions to antigenic components of the streptococcus, which also cross-react with human tissues, such as the heart, valve structures, joint tissues and neuronal antigens. Therefore, it can be complicated by carditis that leaves permanent consequences. It generally occurs in children and adolescents. The prognosis contains serious reservations, especially in complicated cases of rheumatic carditis.

RHEUMATOID POLYARTHRITIS

It is more common in women than in men, and is characterized by polyarticular inflammation that evolves through outbreaks separated by periods of remission. It is autoimmune in nature, although the etiopathogenesis is unknown. May present rheumatoid factor. The prognosis can be serious.

SECONDARY CAUSE ARTHROPATHIES

Arthropathies of secondary cause are called connective tissue pathologies. They are associated with lupus erythematosus, scleroderma, polymyositis, etc. Arthropathies secondary to inflammatory bowel diseases are also described. In this group we must also consider metabolic arthropathies, such as gout.

SYMPTOMATOLOGY

Joint inflammation syndrome can affect several joints at the same time (polyarticular or oligoarticular), either unilaterally or bilaterally, as occurs in acute or chronic polyarthritis; or affect a particular joint, constituting a monoarticular inflammation, very common in trauma or induced infections. The soft parts of the joint present the usual signs of any inflammation, which are: pain, as the most relevant symptom, coming mostly from those points where the synovium is more superficial or presents folds. It increases when pressure is placed on the joint or when movements are attempted. The onset can be abrupt, intense in acute cases, and lead to a certain degree of stiffness in the joint. The muscles close to the joint feel flaccid, while in those farther away, defensive contractions of an antalgic nature occur. When rigidity persists for prolonged periods of time, it can lead to disability in the patient.

On other occasions the pain begins in a latent and insidious form, reducing to simple arthralgias, as occurs in chronic conditions, although the appearance of exacerbation crises is possible.

The increase in volume of the joint (tumor) is a constant sign caused by the exudation of fluid rich in proteins, typical of any inflammatory process.

A "spontaneous painful swelling with redness (redness) and increase in local temperature (heat) occurs. The redness may be absent. Inside the joint there may be an effusion, which may be hydrarthrosis, when the fluid is clear and contains polynuclear cells, henmarthrosis, if the fluid contains blood, and pyoarthrosis, when it is purulent. The effusion is sometimes small in quantity, but when it is abundant it shows prominences in the sites where depressions normally appear, the tissue presenting a certain tension on palpation according to the amount of fluid present. When the tension is not great, the fluid can move from one part of the joint to another. In reality, the nature of the effusion can only be suspected during the clinical examination; if there is little pain, it more commonly corresponds to a hydrarthrosis; if a certain degree of intra-articular crepitation is observed on palpation, the finding is more consistent with a hemarthrosis; if there is fever and the pain is intense, pyoarthrosis should be considered. Only the extraction by puncture of the fluid can establish its true nature.

There are commonly general signs of infection such as: fever, tachycardia and accelerated erythrocyte sedimentation rate. Acute cases are accompanied by leukocytosis with a predominance of polynuclear cells with a shift to the left; On the contrary, in chronic conditions what is seen is a relative lymphocytosis.

Superficial lymphadenopathy is palpable in 30% of cases, the lymph nodes appear hypertrophied, not

They are larger than the size of an almond, are not painful and never ooze.

POSITIVE AND ETIOLOGICAL DIAGNOSIS

To determine the cause of joint inflammation, it is necessary to not only assess the local symptoms, but also the general manifestations that the patient presents: infectious arthritis sets in early, accompanied by irregular fever with high elevations, preceded by chills, leukocytosis, neutrophilia and increased erythrocyte sedimentation rate. The joint is the seat of acute fluxionary inflammation that frequently extends to several joints.

The pain is severe, the swelling is large, the skin is red in the superficial joints, the joint stiffness is very marked and the general condition is poor. The joint puncture presents a creamy, cloudy liquid that contains between 50,000 and 100, 000 cells per cubic millimeter. Almost all cells are polynuclear with signs of alterations. The culture allows the germ that causes the infection to be established.

When its origin is gonococcal, it is associated with the presence of gonorrhea, but the effusion is scarce, not abundant, with marked signs of periarthrosis and great joint stiffness.

Tuberculous arthritis generally appears after trauma, sometimes minor; In its beginnings it appears to be a moderate and chronic inflammation, with pain, functional limitation, swelling and intra-articular exudation.

The pain is more intense at night and is sometimes located at a distance; For example, in vertebral tuberculosis the hip may hurt and in tuberculosis of the hip, the knee. The synovial membrane thickens extraordinarily and in the joint puncture an opaline or clear fluid with no more than 20,000 cells per cubic millimeter is extracted. The tuberculin skin test allows the diagnosis to be specified, but the culture is the essential element.

Infectious rheumatism, particularly acute articular rheumatism, is observed in children and adolescents; As already noted, it is generally preceded by streptococcal pharyngitis; Acute polyarthritis is mobile and heals completely. Its main associated manifestations are constant and high fever of 39 °C, tachycardia, accelerated erythrocyte sedimentation rate, carditis with dull tones and the appearance of a murmur, which when diastolic is not open to discussion and reflects a lesion of the aortic signmoids, but in cases of systolic murmurs it becomes more difficult to attribute it to the rheumatic process, especially if the patient is not in full crisis, since the Murmurs of this type are frequent in normal subjects.

Rheumatoid polyarthritis is usually polyarticular and mainly affects the joints of the limbs, generally symmetrically. It can lead to cartilage and bone destruction, retractions or, on the contrary, relaxation of the ligaments. All of these complications are a source of ankylosis, deformations and joint dislocations that lead to definitive functional impotence.

Allergic arthritis, frequently related to injection of blood serum or transfusions, is accompanied by great pain, hydrarthrosis and high temperature. It resolves spontaneously in a few hours or days.

Finally, gouty arthritis shows a predilection for the metatarsophalangeal joint of the big toe and is accompanied by hyperuricemia.

SACROLUMBALGIA

This condition, which constitutes a syndrome, is caused by the deterioration of the intervertebral discs, generally due to aging and sometimes due to trauma. It usually manifests itself after the age of 30, since the degenerative changes of the disc are very early. Disc deterioration may not be appreciated radiologically, although some degree of bone crushing with or without anterolateral osteophytosis is frequently noted. The presence of degenerative disc disorders, which are very common after the age of 40, is not always accompanied by symptoms; They can be seen radiologically, without the wearer reporting any discomfort.

Low back pain generally corresponds to the deterioration of the last two lumbar discs L4, L5 as well as the first sacrum S1. The main lesions are the transformation of the annulus fibrosus and the collapse of the nucleus pulposus. There are also other conditions that can cause low back pain (fig. 32.1).

Anatomophysiological count of the intervertebral disc

As explained in Chapter 7, the disc is formed by a ring of fibrocartilaginous structure. It is very thick in front and thins laterally, which makes the disc less resistant in its posterior portion. It is inserted above and below the vertebral edges. The disc is covered in front and laterally by the anterior longitudinal ligament that reinforces it, although it leaves small interligamentous areas where anterolateral osteophytosis develops. Behind, the disc has the posterior longitudinal ligament for its coverage, this is very thin and richly innervated by the sinuvertebral nerve, which makes it very sensitive and painful to destruction. In the central part of the disk is the nucleus

AC LC LO R NAL I Blockage of urinary excretion causes acute hydronephrosis Referred pain to the lumbosacral region results from distention of the renal pelvis and capsule Many other kidney disorders also cause lumbosacral pain

B MT STAS SV RT BRAL Sciatica
It is a common manifestation of vertebral metastases, sciatic pain in a middle-aged person should suggest a malignant tumor.

C OST OPOROS Comminuted fractures due to compression or crushing of a vertebra can occur spontaneously or be due to slight trauma, bone pain is frequently diffuse.

DT MOR D LA M D LA SP NAL Generally
The location of the tumor can be determined by the level of pain and the distribution of muscle weakness and anesthesia.

H RN AD D SCO NT RV RT BRAL The hernia occurs most commonly in the region of the fifth lumbar vertebra as a consequence of a sudden rotation movement, a fall, or forced hyperextension of the trunk.

FDF CTOS CONG N TOS I pain produced by partial sacralization of the fifth lumbar vertebra, is due to the presence of an inflammatory process of amphiarthrosis, complete sacralization is asymptomatic

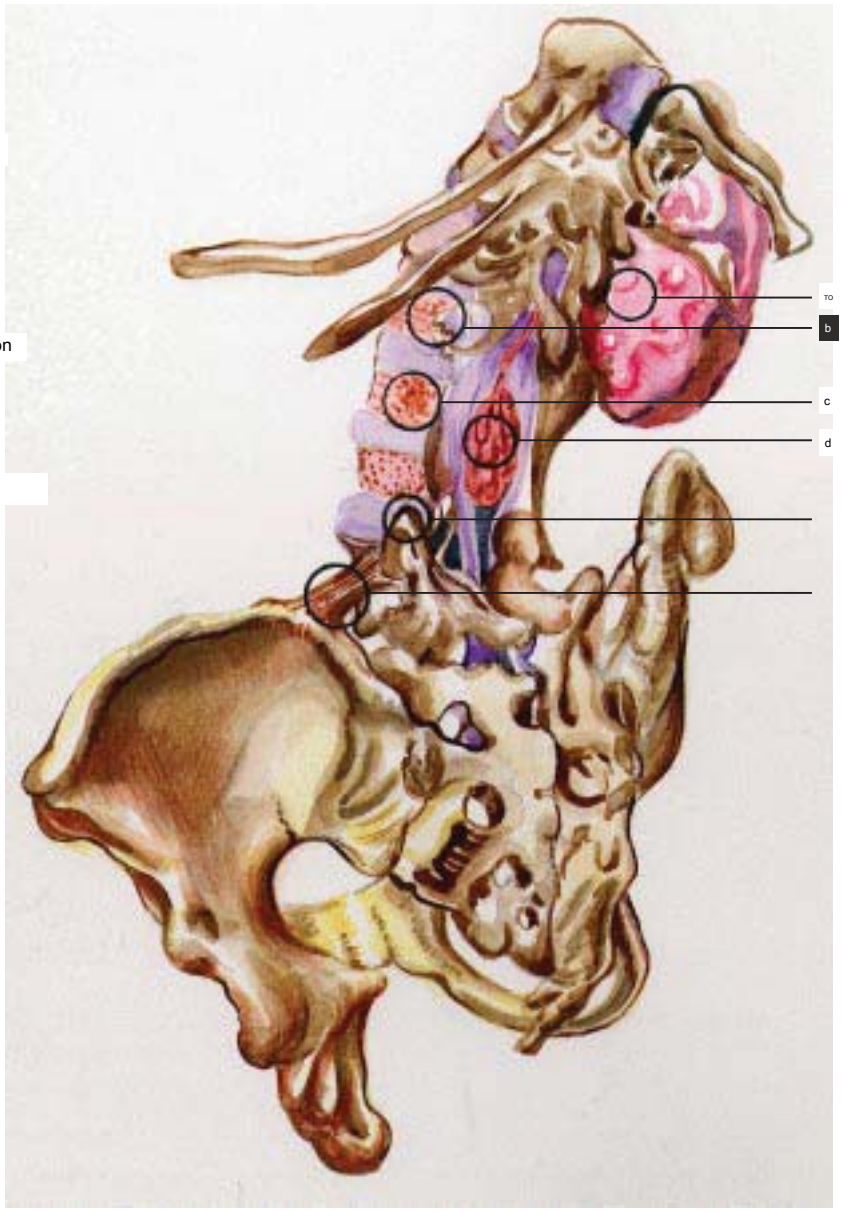


Fig. 32.1 Most frequent causes of low back pain.

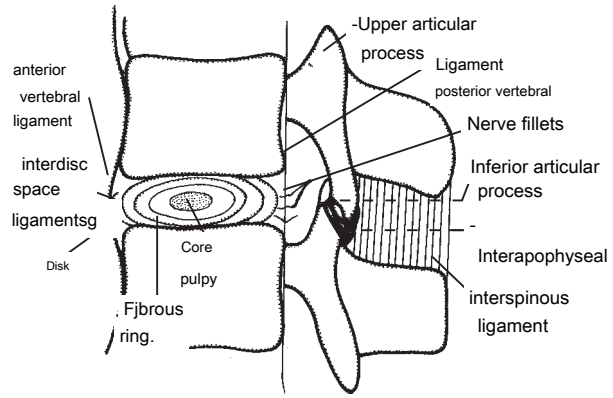


Fig. 32.2 Means of union of the vertebrae.

pulpy, which has the appearance of a whitish and homogeneous jelly. At the lumbar level, the nucleus is located between the middle third and the posterior third of the disc. The vertebrae move around the nucleus, transmitting the forces exerted on it to the rest of the annulus fibrosus, whose elasticity cushions them (fig. 32.2).

Syndromog nesis or pathophysiology

The deterioration is characterized by the thinning of the annulus and the sinking of the disc or nucleus pulposus. If this moves towards the posterior cleft of the annulus whose ligamentous reinforcement is slight, generally very painful clinical manifestations occur (Fig. 32.3). The most frequent pathological expression of posterior nuclear lesions consists of three clinical manifestations:

- Acute low back pain.
- Chronic low back pain.
-Common vertebral sciatica.

ACUTE SACROLUMBALGIA

It is characterized by the compression of the disc annulus and the collapse of the nucleus pulposus, which moves towards the posterior groove of the annulus, giving rise to the clinical manifestations. This picture is abruptly established

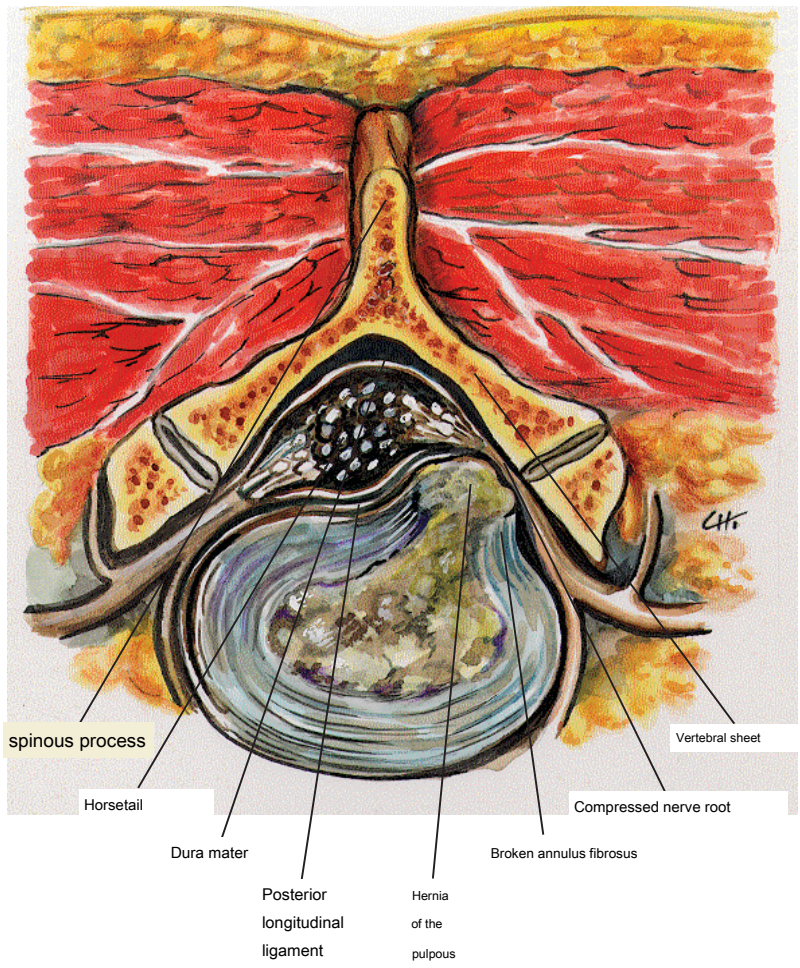


Fig. 32.3 ernia of the pulpous disc.

usually after heavy lifting or a straightening or sudden twisting movement of the torso.

Symptoms

After a heavy lift, the patient suddenly complains of a crunch in the lumbar region accompanied by violent pain that prevents him from standing up completely, sometimes he collapses and requires being lifted and transferred to bed. The pain is located in the lumbosacral region, increasing with effort: coughing, sneezing, defecation, etc.; Its intensity is variable although it is frequently very high. If he is able to walk, he does so painfully and with difficulties. The lumbar spine remains rigid; vertebral static is altered; Defense symptoms may appear characterized by an antalgic position with flattening of the lumbar lordosis and the patient even adopts the opposite position of lumbar kyphosis, inclination of the torso and scoliosis. This antalgic posture that the patient spontaneously adopts is irreducible,

The radiological examination may be negative, although occasionally a crushed lumbosacral disc is seen with or without anterolateral osteophytosis or impingement located at the level of the disc.

The evolution usually lasts 5 6 6 days; the pain gradually disappears; Sometimes the discomfort lasts for several weeks. Although the crisis may occur only once, it frequently occurs at more or less long intervals. It is also possible that other disc deterioration disorders may appear, such as sciatica or chronic low back pain.

CHRONIC LUMBALGIA

It is also due to the deterioration of the lumbosacral discs. It occurs between 30 and 60 years of age and usually the patient reports having suffered from one or several acute crises, although there are cases in which the onset of the condition is insidious in a subject who has never suffered from acute low back pain or sciatica.

Symptoms

The pain is located at a low lumbar level with irradiation to the sacrum and buttocks, its intensity is moderate and increases with some efforts such as loading or flexion and extension movements of the trunk. It is also affected by prolonged standing or sitting for long periods of time, as occurs when traveling long distances. Although rest relieves it, the patient reports upon waking up in the morning a sensation of painful lumbar tightness that disappears little by little, only to reappear again with exercise. Lumbar stiffness is discrete or may not be appreciated. The strong pressure in the inter-pineous joints of L4-S1 can be painful, rarely the symptom exceeds this vertebral height.

Radiographically, disc alterations can be seen: crushing, anterolateral osteophytosis, small posterior displacements of the vertebrae (retrolisthesis). The alteration may be localized to one segment or extend to several, which is the most common. The evolution lasts for several years with remissions and sporadic exacerbations, the latter of the type of acute low back pain or sciatica pain. It can make it difficult to exercise and certain activities that require carrying weights, generally disappearing over a period of 5-10 years.

COMMON VERTEBRAL CIATALGIA

It corresponds to the posterolateral lumbosacral disc herniation. The pain affects the lower limb following the territory of L5 or S1. It is common after the age of 30, like other forms of low back pain, it can occur suddenly after a sudden effort or movement, although sometimes it develops progressively after a heavy lifting effort.

Symptoms

The pain is unilateral and runs through the entire lower extremity; Exceptionally, it is bilateral, although on these occasions it has predominance on one side or the other. The symptoms correspond to the level of discoradicular compression:. This can occur at the level of the L4-L5 discs that irritate the L5 root, or L5-S1 in which case the irritation is on the S1 root, therefore the pain is monoradicular and its characteristics and topography are different.

When it comes to L5 sciatica, the pain is postero-external in the thigh, external in the leg and passes over the external malleolus to cross the dorsum of the foot and end in the big and second toes. The neurological examination shows the existence of a normal Achilles reflex, there is usually hypoesthesia of the painful territory, especially in the dorsum of the foot and big toe. There is also a decrease in muscle strength of the foot elevators, lateral peroneal muscles and extensor hallucis. Usually the patient cannot stand on the heel with the toe raised on the affected side, which constitutes the heel sign.

When it comes to sciatica SI, the pain is posterior in the thigh and leg, passing through the heel to the outer edge of the foot to end in the last three fingers.

The neurological examination shows a marked decrease or disappearance of the Achilles reflex; There is more or less important hypoesthesia and of variable extension over the painful territory, especially on the external edge of the foot and in the last three toes: the

muscle strength in the posterior area of the leg, which prevents the patient from standing on tiptoe on the side of the sciatica, this constitutes the toe sign.

In both cases the pain is variable in intensity, but there are times when it is unbearable and causes total insomnia (hyperalgesic sciatica). Bed rest calms you, although the first days of lying down are painful. Elevation of the lower extremity in extension with the patient lying down ceases quickly due to exacerbation of pain; It is the so-called Lasègue sign.

On examination of the spine, a certain degree of rigidity is observed with the adoption of antalgic positions similar to that of acute low back pain. This antalgic attitude is irreducible; any movement to prevent it causes intensification of the pain. Pressure in the paraspinous region from L4 to S1, according to the particular type of sciatica in question, produces radiation of pain towards the lower extremity following the corresponding and already described path that constitutes the sign of Seze's ringing.

The radiographic examination may be normal, although alterations similar to those described for low back pain are sometimes seen.

The evolution is generally rapid and the condition disappears in a few weeks. Sometimes sciatica is resistant to all treatment and when it persists for three or more months it is necessary to consider the possibility of surgical treatment.

COMPARTMENT SYNDROME

Compartment syndrome occurs when high-pressure fluid accumulates within a space closed by fascia (muscle compartment), decreasing capillary perfusion below the level necessary for tissue viability.

The main causes of compartment syndrome are three:

Increased fluid accumulation.

- Decrease in volume.

-Constriction of the compartment and limitation of volume expansion, secondary to extensive compression

terna.

Although compartment syndrome most often develops in the four compartments of the leg, it can also occur in the forearm, hand, upper arm, shoulder, foot, thigh, buttocks, and back.

These syndromes are rarely diagnosed and that is why we are forced to make a short introduction.

tion for better understanding, referring to the leg where they are most frequent.

The fascia of the thigh, fascia lata, continues into the leg where it is called the leg or crural fascia.

Deep extensions of this fascia form the septa: anterior intermuscular, posterior intermuscular and transverse intermuscular. These septa and the interosseous membrane (tibia-fibula) make up the so-called compartments of the leg.

They are:

Previous compartment.

-Side compartment.

-Deep rear compartment.

-Shallow rear compartment.

Each compartment houses certain muscles, arterial and venous branches, and nerves:

1. Anterior compartment; extensor muscles: tibialis anterior, extensor of the big toe, extensor digitorum longus and third peroneus, which extend the toes and dorsally flex the foot; anterior tibial arteries and veins and deep peroneal nerve.
2. Lateral compartment, peroneal muscles and superficial peroneal nerve.
3. Deep posterior compartment, deep flexor muscles: flexor digitorum longus, tibialis posterior, flexor hallucis longus, popliteus, posterior tibial arteries and veins, peroneal artery and tibial nerve.

4. Superficial posterior compartment, superficial flexor muscles, soleus, gastrocnemius, arteries, veins and nerves, branches of those previously mentioned.

It can now be better understood why different syndromes occur acutely or chronically in any of the compartments, in this case the legs.

ACUTE COMPARTMENT SYNDROME

Syndromography

Clinic

The most important symptom of impending compartment syndrome is disproportionate pain in relation to the initial injury. However, pain may be absent if there is also a disorder of the central or peripheral nervous system.

To remember the initial symptoms, it is useful to use the six P' s rule of compartment syndrome:

1. Pressure. It is the earliest finding in a swollen and tense compartment on palpation.

2. Pain on extension (Pain). Passive movement of the fingers can cause pain in the ischemic muscles involved. The subjective aspect of the patient must be taken into account.

3. Paresis (Paresis). Muscle weakness may be a consequence of primary nerve involvement, muscle ischemia, or pain defense.
4. Paresthesia (Paresthesia). The most reliable physical finding in a conscious and cooperative patient is sensory deficit. Initially, sensory detection manifests as paresthesia, but if treatment is delayed, it can progress to hyperesthesia and anesthesia. Careful exploration of sensitivity helps determine which compartments are involved.
5. Pulses present (Pulse). If there is no injury or severe arterial disease, peripheral pulses are palpable and capillary refill is generally preserved.

6. Pink color.

paraclinical examinations

- Needle technique.

-Wick catheter technique.

-Slit catheter technique.

All three allow the measurement of intracompartmental pressure that may be necessary in three groups of patients:

1. Patients who are uncooperative, untrustworthy, intoxicated by alcohol or drugs and in children.
2. Patients who are unresponsive due to head trauma or drug overdose.
3. Patients with associated neurovascular lesions, or neuropraxia due to associated nerve deficit.

Syndromog nesis

The most frequent causes of this syndrome are: fractures, vascular injury with intracommunal hemorrhage, timental, muscle swelling due to overexertion, exogenous fluid infiltration and others.

ANTERIOR TIBIAL COMPARTMENT SYNDROME

Synonymy

Ischemic necrosis syndrome of the peroneal muscles and peroneal compartment syndrome.

Clinical

Syndromography

This syndrome can appear in one or both limbs, it occurs after prolonged and continuous exercise. The patient reports tiredness of the legs, then edema and severe pain appear, which does not disappear with elevation of the legs or heat.

There is loss of dorsiflexion and inversion. Any movement causes pain.

A firm consistency is felt on palpation of the anterolateral portion of the leg, as well as the absence of a pedis and posterior tibial pulse.

Anesthesia is noted in a triangular area on the dorsum of the foot, at the base of the first and second toes.

In chronic cases, recurrent pain appears after any effort and is only relieved with surgical decompression of the anterior compartment, through fasciotomy. In acute cases, this emergency intervention must be performed to avoid irreversible muscle changes.

paraclinical examinations

Radiology. To exclude trauma.

Syndromog nesis

It is caused by a decrease in blood flow to the muscles after severe exercise and is caused by ischemic swelling of the muscle within its tight fascial compartment.

RESPIRATORY SYSTEM. ANAMNESIC DATA AND MAIN SYMPTOMS

33

The clinical history data collected by the interrogation of patients with the respiratory system is of great importance, for which reason it should always be carried out with the greatest care and in the most complete manner possible, following the great general rules that we learned in General Semiology for its practice.

Many respiratory disorders will be known thanks to the interrogation, thus corroborating Sergent's statement, already mentioned, that each method has its limit of sensitivity. It will only be through the interrogation that we will know about the subjective symptoms that accompany respiratory diseases (pain, anorexia, weakness, etc.).

It will also be through the interrogation that we will learn of the existence of true respiratory syndromes, of the greatest diagnostic importance, which, characterized by appearing and disappearing without leaving traces that can be collected by physical examination, can only be discovered through the patient's oral history. Such is the case, for example, of asthma attacks, as noisy and abundant in symptoms at the time of their development, as they are silent once they have ended.

We will study the semiological value of the data obtained by the interrogation, which in respiratory pathology, as in any other, will always precede the physical examination, in the same order in which they appear noted in the clinical history model, starting with the personal identity data.

PERSONAL IDENTITY DATA

We already said in General Semiology that the first group of data: name, nationality, etc., present almost exclusively administrative and identification interest, and that naturalness, important in Semiology, is studied with provenance. We continue the same behavior here.

PROFESSION

In many cases it acquires diagnostic interest, clarifying, above all, the etiology of the respiratory process being studied. We already mentioned in General Semiology pneumoconiosis, that is, the lung inflammatory processes determined by the inhalation of dusts, more or less irritating, during work (anthracosis, silicosis, calicosis). End-

Finally, it should be remembered that several observers have attributed some intervention in the genesis of pulmonary emphysema to certain professions that require respiratory effort (musicians who use wind instruments and glass workers, among others).

AGE

It is also important, both from the diagnostic point of view and from the point of view of the evolution of the respiratory disease. Certain diseases, such as whooping cough, are seen much more frequently during childhood; The same happens with measles bronchopneumonia. Childhood tuberculosis has special characteristics that are its own. In countries where tuberculosis exists, it is in children that the manifestations of primary tuberculous infection are preferably observed. Nodal involvement is also a clinical fact that stands out in childhood tuberculosis (tracheobronchial lymphadenopathy and others). In adolescence, the clinical symptoms of tuberculosis reinfection are observed more frequently.

In the elderly, bronchopneumonia and pneumonia acquire extraordinary severity, contrasting with the low intensity of their symptoms (very light or absent fever, little general repercussion at the beginning). In children, the symptoms are loud; The evolution and prognosis are more favorable and some symptoms take on very special aspects, such as the point of abdominal side, which in some cases accompanies childhood pneumonia.

Other bronchopulmonary diseases are more common in adults and the elderly, such as chronic bronchitis, bronchiectasis, pulmonary emphysema and pulmonary sclerosis.

SEX

Its diagnostic interest is much less. It must, however, be remembered that vicariant hemoptysis has been reported in amenorrheic women, which has been responsible for serious and regrettable diagnostic errors. In fact, such vicariant hemoptyses are very often nothing more than true tuberculous hemoptysis, even evolutionary, and amenorrhea is the consequence of tuberculous intoxication. So both symptoms, hemoptysis and amenorrhea, are caused by tuberculosis, and not the amenorrhea caused by hemoptysis. On occasions when hemoptysis coincides with the period, it is bronchial endometriosis, that is, endometrial tissue located ectopically in the bronchus or trachea. In these cases, the interrogation is of inestimable value in the diagnosis.

RA A

It has been mentioned as a factor involved in the etiology of pulmonary tuberculosis. A special predisposition to this disease is attributed to the black race, which also occurs in them with greater severity. It is possible that, more than racial reasons, this weakness in blacks is due to the lack of previous contagion (African blacks) or to poor nutrition and lifestyle, for economic reasons in this case. With vaccination at birth, reactivated later, and current nutrition in our country, no new patients have occurred.

ORIGIN

In many cases it presents great diagnostic interest;

It allows, as we said in General Semiology, to diagnose diseases such as hydatid cyst, based on the notion of its existence in the region or country from which the patient comes. A respiratory syndrome that is reported radiographically by the existence of a circular shadow, drawn in a compass, in one of the lungs, has a high probability of being a hydatid cyst when the patient who has it has been exposed to contagion by residing in an infected region. This disease does not exist in our country.

ANAMNESIS NEXT HISTORY OF THE CURRENT ILLNESS

Of greater semiological importance than personal identity data is the immediate anamnesis, which includes the symptoms of the current illness, essential for the diagnosis of diseases of the respiratory system.

We will find in it the symptoms that reflect the pathological disorders of this system, to which we will dedicate a special chapter: side tip, dyspnea, cough, expectoration, hemoptysis, vomiting and voice modifications.
The interrogation will also collect, at this time, the general symptoms that often accompany lung diseases, and which sometimes constitute the only symptomatic expression of the process.

Start. Through close anamnesis we will also know the mode of onset or initiation of the symptoms that accompany the respiratory disease, and its mode of evolution or course.

There are pathological processes of the system that we study that begin abruptly and suddenly, their symptoms arising in a full state of health, at least apparent. Thus, the pneumonia whose initial symptoms we have mentioned

mentioned above; pulmonary infarction, with its intense, sudden, characteristic pain or side pain, the dyspnea and bloody expectoration that accompany it, spontaneous pneumothorax with its dramatic picture of pain and intense suffocation, with tightness and cough, without expectoration on many occasions; essential asthma; serofibrinous pleurisy; etc Other pleuropulmonary diseases begin, on the contrary, in an insidious, slow, gradual and progressive manner. Such is the case of pulmonary tuberculosis and cancer, for example.

Evolution. When we question the patient about the evolution or course followed by his symptoms, we find that in some cases these have been sustained with the same intensity, more or less, once the peak of the process has been reached (pneumonia, for example); other times the evolution is characterized by being gradual and progressive, gradually increasing the intensity of the symptoms and new ones appearing that complete the symptomatic picture of the disease that causes them (chest, suprascapular or infraclavicular pain of increasing intensity and radiating to the arm, low-grade fever, weakness, cough, small hemoptysis, in lung cancer, for example); Other cases, finally, exhibit an evolution in stages, presenting periods in which the symptoms decrease or disappear completely, only to reappear with equal or greater intensity at other times (essential bronchial asthma, for example).

REMOTE Anamnesis

We will discuss separately the semiological value of the two main parts that comprise it: personal pathological history and family or hereditary history.

Knowledge of the previous illnesses suffered by the patient has great diagnostic value in many pathological processes.

Many infectious diseases are responsible for the appearance of diseases of the respiratory system. Thus, the flu causes bronchopneumonia, the same as measles. Whooping cough can also cause them.

Pneumonia presents among its complications lung abscess and purulent pleurisy, called metapneumonic.

In addition to these various infections, there is interest in the pathological history of lung diseases: phlebitis, which can give rise to pulmonary infarction if an embolus detaches from the inflamed vein: heart disease, especially those that dilate the right cavities, which can also cause pulmonary infarctions; and focal infections, such as apical dental abscesses, tonsillitis, sinusitis, etc., which can produce lung abscesses.

The frequent intervention of an allergic factor in the etiology of some diseases of the respiratory system gives great interest to the finding of allergic manifestations among the patient's personal pathological history. Thus, the existence of urticaria, spasmodic rhinitis, headaches, pruritus and other allergic disorders makes it possible to identify the etiology of an asthmatic syndrome or allergic bronchitis suffered by the patient.

cient.

A surgical operation undergone by the patient explains the cause of an acute pulmonary condition that arose in the postoperative period, for which we are consulted, or whose sequelae we have to treat. Such is the case of bronchopneumonia, atelectasis, or postoperative pulmonary

ratio.

Knowledge of the chest trauma that the patient may have suffered is also important, since they are capable of explaining many respiratory processes. Thus, as a consequence of penetrating wounds to the chest, or trauma that causes rib fractures, the following can be observed: pneumothorax, hemothorax, hemopneumothorax, hemoptysis, etc.

In poisoning, it is interesting to highlight the importance of bronchopulmonary injuries caused by the inhalation of the so-called asphyxiant gases, used in times of war. Among toxic habits, tobacco is responsible for, or entertains, many chronic bronchitis.

Attention will also be paid to the lifestyle, type of diet and environment in which the patient lives. Excesses of all kinds, intense and exhausting work, poor nutrition and overcrowding, typical of other countries, are all factors capable of determining, isolated or associated, serious respiratory disorders, explaining, for example, the appearance of evolutionary tuberculous outbreaks, or causing the disease to explode, either by favoring infection, or, above all, by creating a terrain with less resistance in which it easily develops.

MAIN SYMPTOMS OF RESPIRATORY DISEASES

We will now study the main symptoms with which respiratory conditions manifest; These are: chest pain, dyspnea, cough and expectoration, and especially two of great diagnostic value: hemoptysis and vomiting.

Finally we have the voice modifications, which we will discuss later.

Its presence leads us to perform an examination of the respiratory system, although sometimes they are missing and there are only general or digestive symptoms, as we noted before. Sometimes pleuropulmonary disease is

discovered in the course of a systematic examination, it emerges as a finding, since it is not manifested by any symptoms (latent lung diseases). This proves the need and convenience of performing a complete physical examination of the patient, even when there are no symptoms of certain systems.

TORCH PAINS

Concept

Most pleuropulmonary diseases are accompanied by pain, variable in location, intensity and evolution, which have been studied and described by all authors, especially by the ancient classics that insisted on its smallest characteristics.

There is no type of chest pain in diseases of the respiratory system. Rather, there are different types of pain, both in their clinical expression and in their origin, so their interpretation or diagnostic value is also different (fig. 33.1).

Sometimes they are real painful points located in any region of the chest; Other times the pains have a defined topography and coincide with that of many nerve pathways, corresponding to true neuralgia.

Chest pain can be of two types: side pain and neuralgia: phrenic, intercostal, etc.

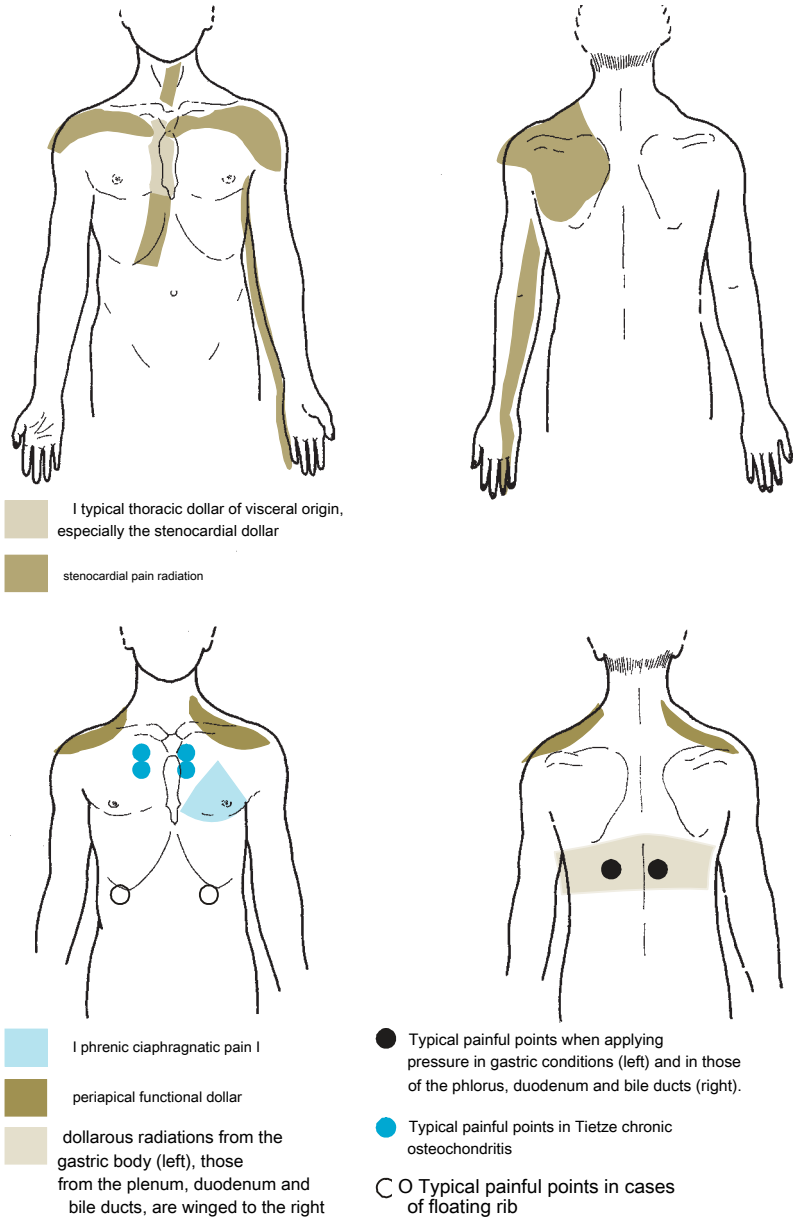

I typical thoracic dollar of visceral origin, especially the stenocardial dollar
stenocardial pain radiation
I phrenic ciaphragnatic pain I
periapical functional dollar
dollarous radiations from the gastric body (left), those from the plenum, duodenum and bile ducts, are winged to the right
Typical painful points when applying pressure in gastric conditions (left) and in those of the phlorus, duodenum and bile ducts (right).
Typical painful points in Tietze chronic osteochondritis
O Typical painful points in cases of floating rib

Fig. 33.1 Typical symptoms of chest pain.

In the first case, we speak of the existence of a point or, better expressed, side pain (point de coté), a very interesting type of these chest pains; In the second case, these are true neuralgias of varied location, due to irritation or injury to the thoracic nerves, such as intercostal neuralgia and phrenic neuralgia.

Semiognesis or pathophysiology

It is unanimously accepted that the parietal pleura and the chest wall, provided with sensory spinal innervation, can be the starting point of painful sensations, and for a long time it has been asserted that all painful manifestations of pleuropulmonary affections are due to the participation of the parietal pleura or the chest wall in the pathological process. Likewise, it has been stated that if this pleuroparietal participation is missing, the pain is absent, in such a way that this symptom would be very important as an element of anatomical diagnosis.

Experimentation in animals and observation in man have shown that irritation or stimulation of the visceral sheet of the pleura and the lung does not produce pain, but instead, it appears as soon as the parietal sheet of the pleura is excited. This has been explained by the lack of spinal sensory nerves in the lung and the visceral pleura that covers it, innervated exclusively by the vegetative nervous system (orthosympathetic and parasympathetic).

Even though clinical observation agrees in many cases with the aforementioned facts, in others, however, it seems to denounce the existence of pain of pulmonary origin. Thus, pain is observed in embolisms located in the center of the lung and in central pneumonia, without the parietal pleura participating in the process. Of greater importance, from this point of view, is the pain produced by the simple distention of the lung stump in pyopneumothoraces, as observed when rapidly aspirating its contents. This distends the collapsed lung, covered by the visceral pleura, but independently of the parietal pleura, despite which the pain appears.

Finally, today two mechanisms of chest pain are accepted: pulmonary pleurovisceral pain (viscerosensory reflex and visceromotor reflex) and pleuroparietal and chest wall pain.

Viscerosensitive reflex. The pleuropulmonary stimulus is transmitted by the vegetative nerves to the posterior horns of the spinal cord, from where this pain is referred to the skin area innervated by the same roots to which this pulmonary segment corresponds. Therefore, in the parietal area to which the pathological process of the lung refers there will be hyperesthesia or hyperalgesia.

Visceromotor reflex. Visceral excitation not only produces pain in the area of the wall innervated by the same spinal cord segment, but also produces muscle contracture at that level. In this case, a visceromotor reflex is established, since the stimulus that starts from the depth and reaches the posterior horn of the cord is reflected to the anterior horn and exits through it, causing contracture of the muscle groups innervated by this part of the anterior horn.

The afferent pathway of the reflex is vegetative and reaches the posterior horn and the efferent pathway corresponds to the spinal motor nerves that leave the anterior horn.

It is important to know these reflexes, since contracture and pain in an area of the chest or abdominal wall allow us to locate the area of lung parenchyma or visceral pleura from which the reflex originates.

Pleuroparietal and chest wall pain. The pain, when the parietal pleura and the chest wall are affected, is due to irritation of the sensory nerves that are distributed there and not, as in the previous case, to the existence of a viscerosensory reflex.

Neuralgias (intercostal, phrenic, branchial) are due to engulfment or compression of the corresponding nerves, due to pleuropulmonary pathological processes.

monars.

clinical study of the side tip

According to Sergent, according to the intensity, duration and form of appearance, the side point can be: acute side point and subacute side point.

Sharp side tip

Concept and semiography

Pain that appears in pleuropulmonary conditions that is located in a specific region of the chest, sharp, intense, continuous, exacerbated by respiratory movements, cough and pressure exerted at its level. When it is very intense it is accompanied by shallow breathing or dyspnea called antalgic polypnea.

Semidiagnosis

Lung conditions:

Pneumonia. Sudden, intense pain in the breast region below the nipple, lasts 2-4 days and disappears if no complications arise. It is accompanied by high fever, cough, expectoration and dyspnea.

Pulmonary embolism. Sudden strong side pain accompanied by dyspnea that sometimes ends in death. If the embolisms are small, the accident passes, accompanied by dark hemoptoic expectoration.

Pleural conditions:

Pleurisy. Less sudden and violent pain than in pulmonary causes, it is located preferably at the base, and extends to the entire pleura. It changes with its location (mediastinal, interlobular, diaphragmatic, etc.).

Spontaneous pneumothorax. Sudden side tip in a stab, tearing pain, which immobilizes the patient and is accompanied by anxiety and dyspnea, which can lead to syncope. After this start, the patient adapts to the new situation.

Subacute side tip

Concept and semiography

Dull, less intense, insidious pain that appears in slowly evolving processes. This side tip is located at precise points on the thorax.

Semidiagnosis

Lung cancer.

The side tip, according to its location, can be:

1. Side tip of the vertex.
2. Cissural side tip, from Sabourín.
3. Side tip of the base.
4. Abdominal side tip.

Each location will have its selective points at a certain level. We will only describe the point of the abdominal side because of its great importance.

Description. Intense abdominal pain that can lead to misdiagnosis with other conditions of the abdomen, particularly appendicitis.

Causes. Pneumonia and other pleuropulmonary processes of the base (generally in children).

clinical study of neuralgia

Other chest pains may also be observed in the course of diseases of the respiratory system. The most important are those due to suffering of the intercostal nerves or the phrenic nerve: intercostal neuralgia and phrenic neuralgia.

Intercostal neuralgia

Concept and semiography

Continuous pain with intense paroxysms along the course of an intercostal nerve. This neuralgia is identified by verifying that the pain appears on palpation of the posterior, lateral and anterior Valleix points, which are the places where the intercostal nerve becomes most superficial.

Semidiagnosis

1. Pleuropulmonary symphyses that encompass the nerve.
2. Lung cancer that can cause this neuralgia through two mechanisms:

a) Invasion of the nerve by the tumor.

b) Spinal metastasis that causes root compression.

3. Neuritis: herpes zoster.

Phrenic neuralgia

Concept and semiography

It is the pain that appears as a consequence of the irritation of the phrenic. Phrenic neuralgia causes intense pain that affects the nerve along its path, with a series of special painful points detailed below:

1. Transverse processes of the fifth cervical vertebra (phrenic emergence).
2. Between the inferior insertion bundles of the sternocleidomastoid.

At this level the phrenic is very superficial, since it rests on the anterior scalenus.

3. Juxtasternal point: internal and anterior extremity of the intercostal spaces at the level of cartilages 5 and 6.
4. Guéneau de Mussy diaphragmatic button. Located by the intersection of two lines, one that extends the left edge of the sternum and another perpendicular to the previous one and which is the extension of the anterior end of the tenth rib.
5. Painful hemibelt at the base of the tőrax 2-4 finger widths above the costal margin, which corresponds to the costal insertions of the diaphragm.

Semidiagnosis

1. Tuberculosis of the vertex with pleuropulmonary symphysis.
2. Tumor processes.
3. Diaphragmatic pleurisy.

It has been observed that in the course of pleuromediastinitis and mediastinal tumors that encompass the phrenic, there is paralysis without pain. This made certain authors think that the phrenic had sensory fibers in the cervical region and only motor fibers in the intrathoracic region and that chest pains not determined by lateral tip were due to intercostal neuralgia and not phrenic neuralgia.

The pain caused in the chest by chronic aortitis, angina pectoris, osteitis, herpes zoster, Pott's disease, tabes, etc. should not be considered phrenic neuralgia.

DYSPNOEA

Concept

Dyspnea means difficult breathing. It comes from dis: difficult; pnein: breathe. It has been aptly defined by Matkins as the conscious and unpleasant sensation of respiratory effort. In addition to this subjective component (difficult breathing), it has another objective, which is the active participation of the accessory muscles of breathing (neck muscles for inspiration and abdominal muscles for expiration) .

semiography

Due to its duration, dyspnea can be acute or chronic and progressive or temporary; Due to its degree or intensity it can be: very light with little discomfort and low respiratory frequency, very intense: it is accompanied by great disorders, anxiety, asphyxia, cyanosis, bradypnea and on other occasions it determines special attitudes such as orthopnea; And due to its appearance, dyspnea can be: slow and gradual or sudden and paroxysmal.

Finally, dyspnea can appear during exertion (exertional dyspnea) or at rest (permanent dyspnea), or suddenly appear generally at night (paroxysmal nocturnal dyspnea).

Subjective phenomena of dyspnea (that is, what the individual with dyspnea feels):

1. Conscious perception of the respiratory act.
2. Shortness of breath, shortness of breath or choking.
3. Weight or oppression in the chest.
4. Throat constriction.

It is logical to think that all subjective symptoms will vary with the intensity and causes of dyspnea, as well as the sensitivity of the patient.

Objective phenomena of dyspnea:

1. Frequency alteration: tachypnea or polypnea and bradypnea.
2. Alteration of breathing times:
   a) Inspiratory dyspnea.
   b) Expiratory dyspnea.
3. Alteration of breathing rhythm:
   a) Cheyne-Stokes.
   b) Kussmaul.
   c) Biot.

Polypnea or tachypnea

Concept and semiography

Increased respiratory rate that can range from 16-20 times per minute to 50 or 60. This tachypnea It is accompanied by a decrease in respiratory amplitude.

Accompanying symptoms are: paleness or cyanosis, chills and signs of suffocation.

Semidiagnosis

Respiratory conditions, circulatory conditions, fever, hemorrhages, emotional crisis, etc.

Bradypnea

Concept

Decreased respiratory rate. Respiration is infrequent and of greater intensity than that corresponding to polypnea.

Semiogenesis or pathophysiology

It is due to obstacles to the entry or exit of air; Therefore, it will be inspiratory dyspnea and expiratory dyspnea.

Inspiratory dyspnea

Inspiratory dyspnea is caused by an obstacle in the upper airways, larynx and trachea, which prevents air from entering the lung.

semiography

Difficult, slow, incomplete inspiration and reversal of respiratory time. Use of accessory muscles of respiration. Trunk backwards due to contraction of the vertebral extensors. Orthopnea in maximum degree of this breathing. The two most important symptoms that accompany inspiratory dyspnea are: cornage or stridor and drawing.

Cornage or stridor: Noisy inspiration when air passes through the narrowed area.

Printing. Paradoxical inspiratory depression of the soft tissues produced by the intrathoracic vacuum that originates when in inspiration the lung does not entirely follow the thoracic cage in its expansion, since the amount of inspired air cannot satisfy this need.
It can be suprasternal or infrasternal, supraclavicular or infraclavicular, or intercostal.

Expiratory dyspnea

Semiogenesis or pathophysiology

1. Loss of lung elasticity.
2. Spasm of the respiratory muscles.
3. Spasm of smooth muscles.
4. Edema and viscous and adherent secretions within the bronchial lumen.

semiography

Great expiratory difficulty that prevents air from escaping from the lung. In this dyspnea it is necessary to use the accessory muscles of expiration, such as: those of the abdominal wall, triangular of the sternum, posterior inferior serratus minor and quadrate of the loins.

In this dyspnea, the patient has great anxiety and cyanosis, and to relieve himself, he compresses the chest with his hands to empty the lung of the air it contains.

Semidiagnosis

Asthma: spasm of the Reisseisen muscles and bronchial obstruction.

Emphysema: loss of lung elasticity.

Cheyne Stokes Breathing

Concept and semiography

Cheyne-Stokes dyspnea consists of the periodic succession of apnea and hyperpnea phases. Although periodic dyspnea surprises the patient awake, generally during the apnea phase he closes his eyes and loses consciousness, in such a way that the patient appears to be dead and, at those moments, it is only the perception of the pulse and the auscultation of the heart sounds that allows us to discard this idea.

After 10-40 s, during which all respiratory movement is suspended, respiratory automatism resumes, but in an irregular manner, beginning with shallow breaths in slow succession, which progressively increase in depth and frequency, until they become of great amplitude, to gradually decrease again, eventually becoming almost imperceptible, and continue with another period of apnea similar to the previous one.

Usually, the hyperpneic phase lasts longer than the apnea period. The total duration of each cycle, which includes both phases, can generally be estimated at 1-3 min. It is common for the succession of each cycle to be done rhythmically; although, on occasion, the duration of the periods of apnea and hyperpnea varies from one cycle to the next (Fig. 33.2).

If, as we have said, during the suspension of respiratory movements the patient is generally unconscious, without responding to external stimuli, during the hyperpneic phase he experiences a certain degree of excitement: he opens his eyes widely, expressing a feeling of anguish, he feels restless and sometimes resumes the conversation that he left on hold during the period of apnea.

It is common to observe constricted pupils in apnea and mydriatic pupils in hyperpnea. Blood pressure can undergo undoubted variations if blood pressure is compared.

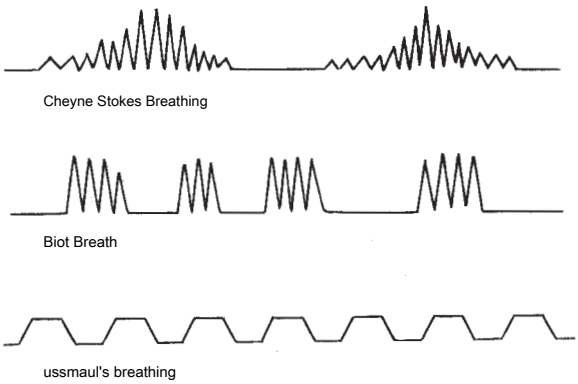


Fig. 33.2 Most common respiratory types.

colds obtained during the hyperpneic phase and those recorded in apneas. The latter are superior. Limón, when measuring systolic and mean pressures by arterial puncture, found figures of 158 mm Hg in hyperpnea and 179 mm Hg in apnea for the maximum, and 120 and 142, respectively, for the minimum.

Semiogenesis or pathophysiology

A general mechanism of production of periodic dyspnea can be established with the facts that we know so far, although we must recognize that there are many points pending to be resolved that leave research open.

Of course we can accept that all cases of periodic breathing, even those we observe in healthy people, are due to a decrease in excitability of the respiratory center. In circulatory patients, this depression is probably caused by a decrease in the supply of CO to the respiratory center. Most cardiovascular patients who exhibit this peculiar form of dyspnea have a decreased partial tension of CO in the blood (13 of the 18 patients studied by Harrison). Those who do not have reduced CO tension in the blood, however, present a reduction in cerebrospinal blood flow, either due to cerebral vascular alterations (atherosclerosis, arterial hypertension) or cardiac alterations (advanced left ventricular failure). That is to say that, in any case, the access of CO to the respiratory center is reduced, either because its content in the blood is lower, or because the blood volume that bathes its neurons decreases. This explains why inhalation of CO often regularizes the breathing of these patients.

Several authors admit that the anoxia that patients with periodic dyspnea may experience explains its production. It is difficult for this to happen, since when arterial oxygen pressure decreases, the respiratory center, far from reducing excitability, becomes hyperexcitable. Furthermore, it is not the most constantly anoxemic heart patients who exhibit dyspnea.

periodic; We have already stated that its occurrence in patients with predominantly right-sided heart failure (mitral, cor pulmonale) is exceptional and that, on the other hand, vascular heart failure with left ventricular failure usually presents it. As we said when describing exertional dyspnea, patients with left ventricular failure rarely have a decreased partial pressure of O2 in the blood and more frequently have a decreased pressure of CO2.

Cardioarterial patients may present another alteration that reduces the flow of blood and, therefore, CO, to the respiratory center. This is hypertension of the cerebrospinal fluid, which is sometimes linked to high blood pressure. Cerebrospinal hypertension can contribute to blocking blood circulation in this territory to a greater or lesser degree. Hence, Uhlenbruck, Fishberg and other researchers have managed to eliminate periodic dyspnea in some vascular patients with hypertension of the cerebrospinal fluid, through lumbar puncture. This also explains the mechanism of production of Cheyne-Stokes dyspnea in patients with central nervous lesions capable of generating intracranial hypertension. In all these cases, it is likely that the decrease in CO access to the respiratory center determines periodic dyspnea, by raising the excitability threshold of its neurons.

On other occasions, a toxin is responsible for the depression of the respiratory center, such as morphine, barbiturates and alcohol, or several toxins, as occurs in uremia or diabetic coma; In these latter cases, the mechanism that produces periodic dyspnea in cardiovascular patients usually also intervenes, to a greater or lesser extent, since frequently these patients are also vascular (hypertensive or atherosclerotic).

In summary, we can say that depression of the respiratory center occurs:

1. Due to decreased CO contribution to the respiratory center river:

   a) Decrease in CO tension in the blood (in most arterial cardiac patients).
   b) Decrease in cerebromeningeal blood flow (vascular alterations, advanced left ventricular failure, cerebrospinal hypertension in patients with the central nervous system and some vascular ones).

2. Due to toxic action with depression of the respiratory center river:

   a) Morphine, barbiturates, alcohol. Diabetic or uremic coma (factor involvement, decreased blood flow, through cerebral vascular lesions).

It now remains for us to explain the determinism of the two phases that constitute each cycle of periodic respiration. It is easily understood that the decrease in excitability of the respiratory center is capable of causing the period of apnea; Now, apnea, as it continues, produces a decrease in blood oxygen tension and an increase in CO2 tension;
When these alterations reach a certain level, higher than the high threshold of excitability of the respiratory center, they again cause the respiratory movements that constitute the hyperpneic phase. The hyperventilation that motivates this period of the cycle increases the O tension again, and progressively decreases the blood CO tension. When these alterations are sufficiently accentuated, the gaseous stimuli of the respiratory center are lower than the excitability threshold of its neurons, so the automatism of the respiratory center is suspended again and the following period of apnea ensues, which repeats the cycle described.

Semidiagnosis

1. Uremic coma.
2. Eat barbiturate.
3. Opiate intoxication.
4. Cerebromeningeal hemorrhage.
5. Meningitis.
6. Heart failure.

Biot Breath

Concept and semiography

There is another variety of periodic dyspnea, described by Biot (see Fig. 33.2), and known by that name, in which all the respirations that follow the period of apnea are rhythmic and of equal amplitude, without the crescendo and decrescendo characteristic of the hyperpneic phase of Cheyne-Stokes respiration.

Semidiagnosis

1. Meningitis.
2. Brain tumors and hemorrhages.

ussmaul's breathing

Concept and semiography

A deep, loud inhalation is followed by a pause;
This is followed by a short exhalation, sometimes with moaning (complaining breathing). Then the expiratory pause and again the noisy inspiration. Kussmaul called it great breathing (see fig. 33.2).

Semidiagnosis

Acidosis, especially in diabetic coma.

General pathophysiology of dyspnea

Previous considerations:

1. The respiratory center or vital node of Flourens, located in the floor of the IV ventricle and responsible for controlling respiratory movements, works automatically (fig. 33.3).

2. The respiratory center receives influences from the brain and nervous and chemical stimuli that guarantee automatism and normal rhythm.
3. Nervous stimuli. The centripetal pathway travels through the pneumogastric tract that comes from the lung where alveolar distention causes the expiratory reflex and vice versa (Hering-Breuer reflex). The centrifugal pathway travels through the phrenic, spinal, and expiratory nerves.

4. Chemical control. Low pH, that is, acidic, stimulates the respiratory center and vice versa, which is why the increase in CO in the blood produces polypnea. Also the decrease in O tension at the level of the respiratory center excites it.
5. The imbalance of these mechanisms will cause dyspnea, either due to alteration of chemical regulation, or due to central or peripheral nervous disturbance.

According to these concepts, the pathogenic causes of dyspnea are:

- Dyspnea due to anoxia.
- Dyspnea due to acidosis.
- Dyspnea due to exaggeration of the Hering-Breuer reflex.

Dyspnea due to organic or functional brain disorders.

Dyspnea due to anoxia

We prefer to call this process anoxia following Van Slyke and not anoxemia as Bancroft calls it, since the 0 deficit is found in all tissues and not only in the blood.

Types of anoxia:

- Anoxic anoxia.
- Anemic anoxia.
- Anoxia due to stasis or circulation.
- Histotoxic anoxia.

Anoxic anoxia. In this type, the O2 tension in the arterial blood is lower than normal. Hemoglobin is not saturated and therefore there is an increase in reduced hemoglobin in arterial blood.

Arterial blood can be poor in O, due to two mechanisms, which are:

1. Little alveolar 0 tension, which is due to:

a) Poverty of O, in the atmosphere (high altitudes, tunnels, etc.).

b) Defective alveolar ventilation, as occurs in the obstruction of air tubes; or due to decreased lung elasticity (emphysema), inability of the lung to hematosis: alveolar-capillary fibrosis.

c) Reduction of large areas of functioning lung parenchyma, as occurs in pneumonia and massive atelectasis, effusions and pneumothorax of a certain intensity.

2. Short circuit, with the corresponding mixture of arterial and venous blood, which can be due to:

a) Persistence of the Botal foramen.
b) Persistence of the Panizza hole.
c) Persistence of the ductus arteriosus.
d) Arteriovenous fistulas.

Anemic anoxia. It is due to a decrease in the oxygenation capacity of hemoglobin, that is, the hemoglobin available to transport O2

This type of anoxia appears when hemoglobin is decreased, as in anemia, when it is combined with carbon oxide, or when it is altered by the action of certain toxic elements that transform it into methemoglobin or sulfohemoglobin.

Stasis or circulatory anoxia. It is due to blood stasis that slows down circulation and produces prolonged contact of blood with tissues and therefore greater loss of O,. This anoxia is accompanied by peripheral cyanosis.

Histotoxic anoxia. It appears in tissue intoxication by cyanides and alcohol, which paralyze the yellow respiratory ferment of Warburg and Christian, preventing the oxidation of cytochrome and thus tissue oxidative processes.

The mechanism that produces the other three causes of dyspnea is not selectively studied, which, as we have mentioned, are: acidosis, alteration of the Hering-Breuer reflex and brain disorders. They can be easily understood by their definition alone.

We are now in a position to apply the pathogenic mechanisms, already studied, of the different respiratory diseases that cause dyspnea:

1. Obstruction of air passages. There is anoxic anoxia due to the low tension of O, which does not arrive in can-

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AF R NTS

MP LSOS N RV OSOS
FR NT S

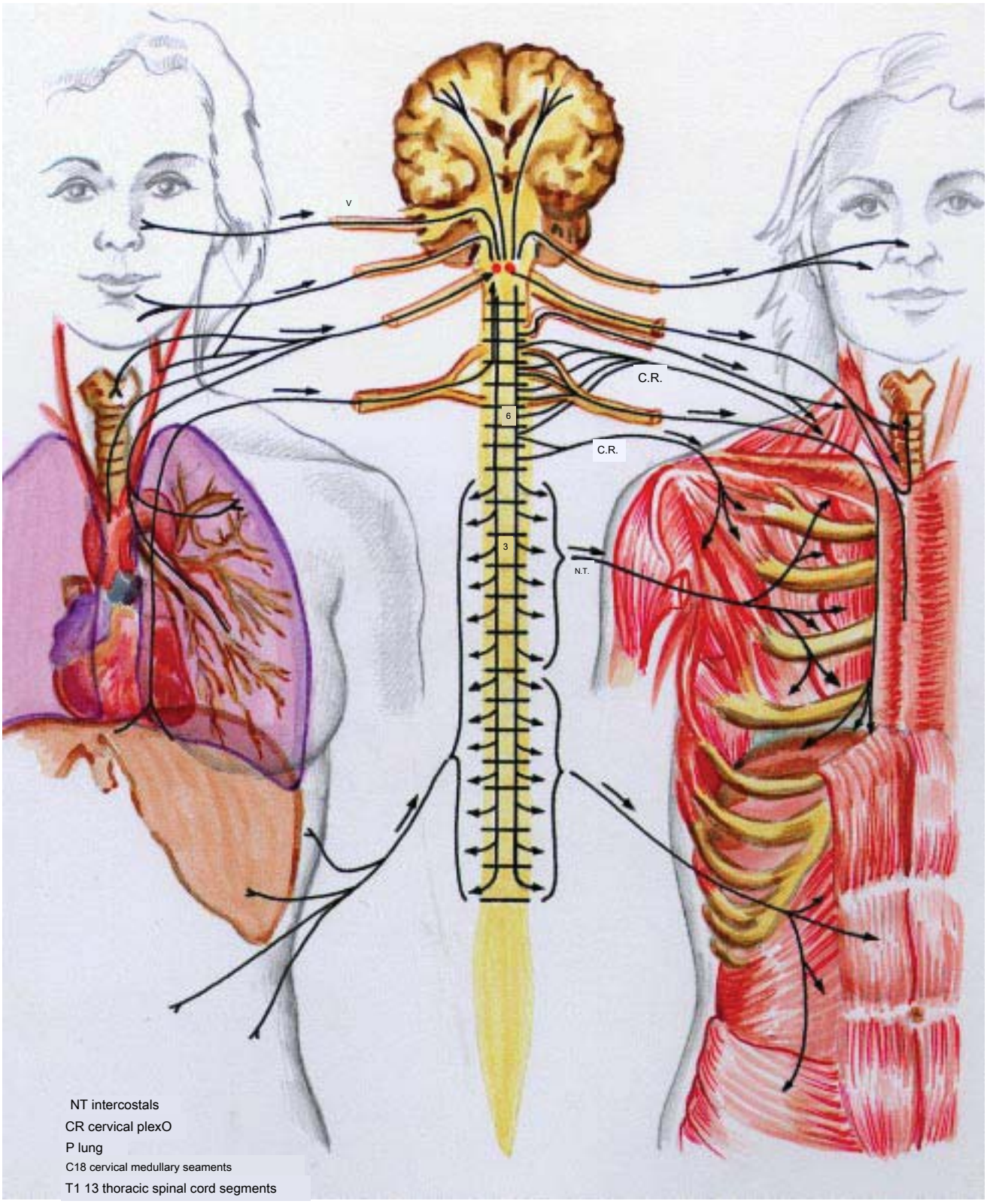


Fig. 33.3 Neuromuscular control of breathing.

adequate amounts to the alveolus and also due to gaseous acidosis caused by the difficulty in expelling CO, which accumulates in the alveolus and therefore in the blood.

2. Pulmonary fibrosis and emphysema. There are the two previous causes, plus an increase in the Hering-Breuer reflex, plus anoxia due to stasis if there is heart failure.
3. Pneumonia, bronchopneumonia and atelectasis. In them there is persistence of blood circulation through a lung incapable of hematosis (anoxic anoxia) and also modification of the Hering-Breuer reflex.

4. Respiratory arrhythmias. They have already been studied in this chapter.

General semidiagnosis of dyspnea

Dyspnea can appear in conditions unrelated to the respiratory system, but its intensity sometimes and the fact that it is sometimes the only symptom of the diseases pleuropulmonary lesions give great diagnostic value to the same further.

We will not study here cardiac dyspnea, for example, which is of great interest; We will only refer to the following:

-Obstruction of air passages.

- Pulmonary processes.

Pleural processes.

Thoracic wall processes.

Obstruction of the air passages A.

Thick passages (larynx, trachea and thick bronchi).

It is an inspiratory dyspnea with cornage and drawing; It may be of sudden onset, as occurs in edema of the glottis or due to inhalation of foreign bodies, or of more or less slow and progressive onset, as occurs in inflammatory laryngeal narrowings: diphtheria, tuberculosis, and in tumor obstructions and compressions.

In the trachea there are the same previous semiological values and in particular the goiter and in the thick bronchi we have the same semiological values.

B. Fine bronchi.

The dyspnea is expiratory with a large amount of wheezing, as occurs in asthma. It is due to spasm of the smooth muscles of the bronchi, to which is added a viscous and adherent secretion on their wall.

Pulmonary processes

In them, the intensity of the injury sometimes does not match the degree of dyspnea:

- Lung infarction (paroxysmal dyspnea). Postoperative atelectasis (paroxysmal dyspnea).
Bronchopneumonia (constant dyspnea since the beginning of the disease).
- Tuberculosis (dyspnea is not always related to the intensity of the injury. Example: granulia).
- Emphysema (expiratory dyspnea).

pleural processes

- Acute pleuritis (side tip with antalgic polypnea).

Pleural effusions (the intensity of dyspnea depends on the amount and speed of installation of the fluid).
- Pneumothorax (sudden dyspnea, resulting in valve pneumothorax, becoming asphyxiating).

Thoracic wall processes

Painful processes that immobilize the wall produce antalgic polypnea, as seen in myositis, rib fractures, etc.

COUGH

Concept

It consists of one or several strong and sudden expirations due to a sudden contraction of the expiratory muscles, initially accompanied by spasms of the constrictors of the glottis, which closes and then opens abruptly under the action of the expiratory force, allowing a characteristic noise to be heard produced by the violent escape of air accumulated under tension in the airways, accompanied or not by secretions or foreign bodies existing in them.

When the cough manages to drag secretions, it is called a wet cough or useful cough, but sometimes it is due to reflex action, as occurs in pleuritis and tracheobronchial lymphadenopathy, and is then called a dry cough.

Semiognesis or pathophysiology

Cough can be voluntary, but it is generally reflex, and therefore consists of (fig. 33.4):

- Starting point.
-Centripetal pathways.
-Cough center.
- Centrifugal routes.
-Causes that irritate the centripetal pathway.
- Response or description of the reflection.

Starting point

True cough zones:

- Interarytenoid space: superior laryngeal nerve.
- Tracheal bifurcation: pneumogastric nerve.

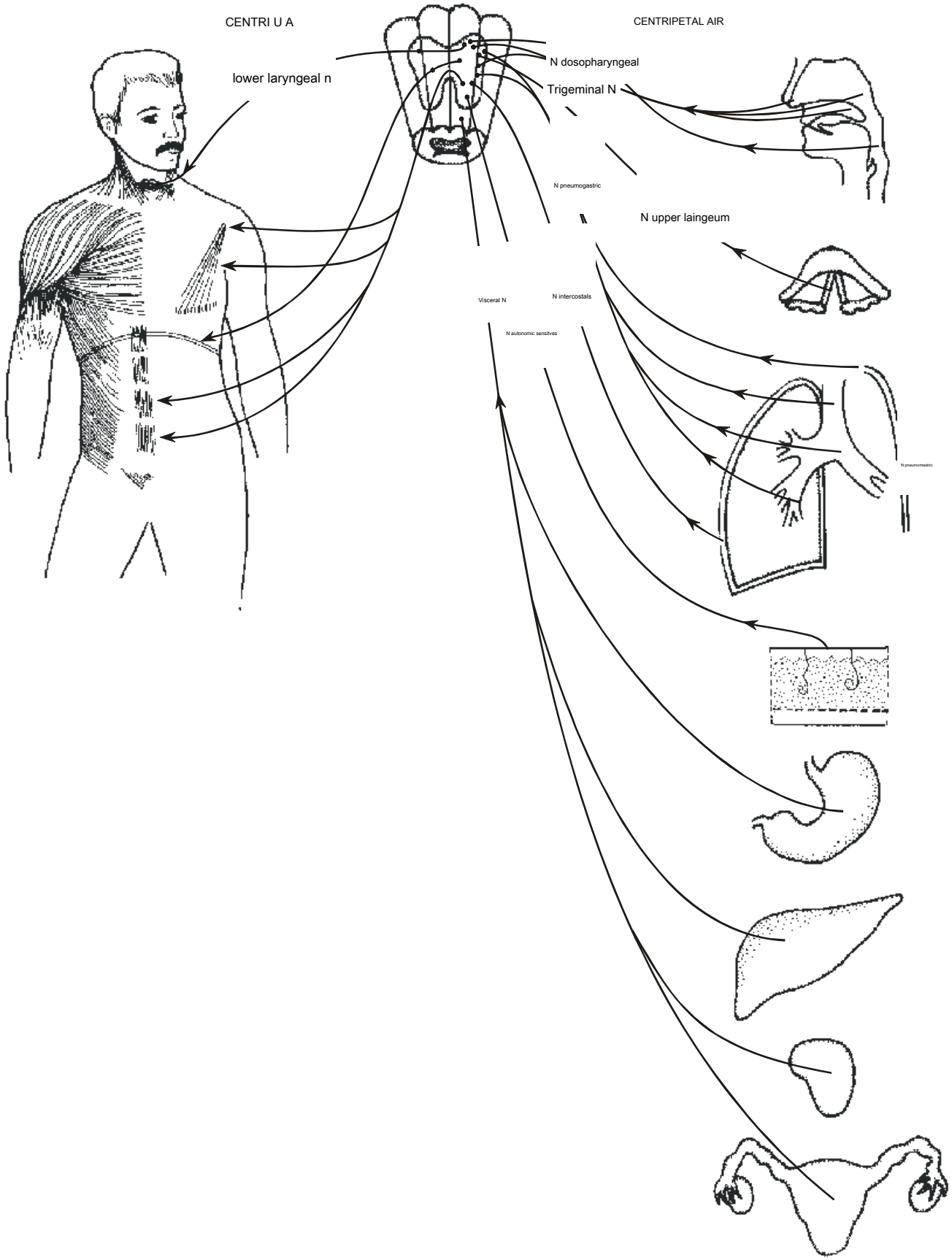


Fig. 33.4 Pathophysiology of cough.

-True vocal cords: superior laryngeal nerve and pneumogastric nerve.

Other starting points:

- Nasal mucosa: nasal cough.
- Tracheal mucosa: tracheal cough.
- Bronchial mucosa: bronchial cough.
- Pleura: pleural cough.
- Pharynx: pharyngeal cough.
- Tonsil: tonsillar cough.

Viscera: visceral cough, depending on whether the starting point of the reflex comes from the esophagus, stomach, intestine, etc.

- Skin: sensitive cough.
- Direct excitation of the pneumogastric: mediastinal tumors that compress it and also in the case of atrial cough due to excitation of the auricular branch of the pneumogastric.

centripetal pathways

- Upper laryngeal: part of the true cough zones.
- Pneumogastric: part of the true cough zones.

ras.

- Trigeminal.
- Glossopharyngeal.
- Sensory nerves of the skin.
- Visceral nerves.

cough center

It lies in the medulla, but some authors locate it at the level of the third ventricle next to the quadrigeminal tubercles.

Centrifugal pathways

- Upper laryngeal.
- Pneumogastric.
- Phrenic.
- Intercostals.
- Spinals.

These expiratory nerves, when excited, contract the muscles, causing coughing.

Causes that irritate the centripetal pathway

Bronchopulmonary secretions.

-Foreign bodies.

In these cases they penetrate and excite the cough areas.

According to Ameville, cough areas are sometimes punctate, therefore, sudden hypertension of a blood vessel near a cough area can cause cough.

Response or description of the reflex

When the center is excited, the response is produced in the following way: sudden inspiration that may be missing, momentary closure of the glottis, sudden expiration that tends to expel air, which is prevented by the glottis being closed. Noticeable increase in internal pressure with dilation of the trachea, violent contraction of the expiratory muscles. Opening of the glottis. Closing of the nasal passages due to the soft palate that rises. Expulsion of air that was lodged in the airway, violently through the mouth. Drag of foreign bodies and secretions. Sometimes accompanying accidents due to increased pressure.

semiography

- Dampness and dryness of cough.
- Intensity and frequency of cough.
- Cough rhythm.
- Tonality and timbre of the cough.
- Emetic cough.

Dampness and dryness of cough

Wet cough is due to the mobilization of secretions accumulated in the airways and is recognized by a special superimposed noise and is almost always accompanied by expectoration. It is called a pleasant cough.

Dry cough appears when there are no secretions and if there are they are viscous, adherent and cannot be mobilized; Then you hear a characteristic noise. Dry cough is more annoying and for this reason it is called unpleasant cough.

Semidiagnosis

The causes of dry cough are:

1. Processes that determine the cough reflex without secretions in the respiratory tree. This cough appears in pleuritis and in extrarespiratory conditions such as gastric cough, auricular cough, uterine cough, etc.

2. Inflammatory conditions of the respiratory system such as: laryngitis, tracheitis, bronchitis, pneumonitis, etc., before secretions appear.

The causes of wet cough are:

1. Pulmonary processes with secretion that must be expelled as occurs in pulmonary suppurations, chronic bronchitis, etc.

intensity and frequency of cough

The intensity and frequency of cough depends on:

Degree of irritability of the patient.

-Amount of expectoration.

Inflammation or irritation of cough areas.

- Causal disease.

According to the factors indicated, cough can be:

scarce, not very intense, frequent, persistent, quibbly, emetic and belching.

Semidiagnosis

The causes that determine intense and frequent cough are:

1. Laryngitis.
2. Tracheiis.
3. Pneumonitis.
4. Abundant secretion in pulmonary suppurations.
5. Pleurisies at the beginning.

Cough rhythm

Cough can be:

a) Dry, light and continuous: as observed in beginning pulmonary tuberculosis.

b) Strong, doglike, repeated at periodic intervals: as observed in mediastinal processes.

c) Quintose: made up of several sudden expiratory jerks with deep inspirations interspersed.
Sometimes loud and sibilant, reminiscent of the rooster's crow, called repetitions or reprises. The fundamental cause of quintase cough is whooping cough in its second period. It has this name because each access has five reprises, or according to other authors because the accesses appear every 5 hours.

In whooping cough, the spasm of the glottis is intense, with cyanosis, although death from asphyxiation rarely occurs.

The crisis ends with violent and scant expulsion of hyaline mucus.

In mediastinal processes, irritation of the pneumogastric tract due to compression exerted by tumors or inflammation gives rise to a cough of less frequency and duration, without reprises or hyaline

Tonality and timbre of cough

a) Bitonal cough; It presents marked changes in its tone due to disturbances of the vocal cords, almost always unilateral paralysis. Example: aortic aneurysms, tumors that compress the recurrent, etc.

b) Hoarse cough; particular timbre sometimes compared to the barking of a dog (barking cough). It appears in laryngitis, especially in diphtheria cough or croup.

c) Hoarse or hoarse cough; dull or veiled ringing. It appears in tuberculous laryngitis, or in cancer, or by paralysis of the vocal cords. The latter is due to poor closure of the glottis. It is also observed in weakened or paralyzed patients without the strength to produce a cough of sufficient intensity.

Emetic cough

It is a cough that causes vomiting. It is the so-called Morton's cough.

Semiogenesis or pathophysiology

The excitability of the gastric mucosa facilitates dilation of the stomach when eating food. The dilated stomach rejects the diaphragm and compresses the base of the left lung. This compression by itself or by mobilization of secretions causes a cough. This cough causes vomiting due to the pressure that the sudden movements of the diaphragm and expiratory abdominal muscles exert on the occupied and dilated stomach. It has also been pointed out that vomiting is produced by irradiation to the center of the stimuli that reach the cough center. This irradiation is due to the proximity that exists between both centers.

This emetic cough has made Peter and Marfan point out that the tubercular patient coughs because he eats and vomits because cough.

The prognosis of this cough, especially in tuberculosis, is very serious, due to the nutritional deficiency with weight loss and cachexia that it causes.

Semidiagnosis

1. Whooping cough.
2. Mediastinal conditions that compress the pneumogastric.
3. Evolutive fibrocaseous pulmonary tuberculosis with a serious prognosis.

Consequences of cough

1. Increased intrathoracic pressure. This hinders the return of venous blood to the right atrium, with venous stasis in the head, evidenced by engorgement of the neck veins and cyanosis, conjunctival hemorrhages, epistaxis, cerebral hemorrhages (rare).
Small circulation disorders. Pleuropulmonary accidents.

a) Spontaneous pneumothorax.

b) Hernia and rupture of the lung with subcutaneous emphysema neo concomitant.

2. Increased intra-abdominal pressure. The compression exerted on the abdominal viscera by the expiratory muscles of the belly wall causes vomiting in the so-called emetic cough, and also the involuntary emission of urine and feces.

EXPECTORATION

Concept

It is the expulsion through the mouth and through coughing of the secretions accumulated in the respiratory tree.

Expectoration can present very particular characteristics in certain respiratory tract conditions and constitute a valuable diagnostic aid, in these cases, for example, rusty sputum in pneumonia.

Expectoration is made up of different components: products resulting from necrosis, blood, pus, caseum and, as a common diluent of the same, the hypersecretion of the mucous glands of the tracheobronchial lining.

Semiography and semidiagnosis

The most important characteristics of expectoration are:

-Quantity or abundance.

Viscosity or consistency.

-Color and transparency.

- Subjective smell and taste.

The amount of expectoration depends on the nature of the lung lesions, as well as the strength of the patient's cough. The suspension of expectoration in a cachectic or weakened patient is a prognostic symptom of severity, therefore, it expresses the degree of exhaustion of the patient.

At the beginning of the pulmonary symptoms, the amount of sputum may be small and even in certain conditions such as pleuritis, it may be completely absent, but as the condition progresses, the associated process, generally inflammatory, causes a great increase in expectoration, which in some diseases undergoes modifications in the day, and it is possible to speak of a schedule as occurs in bronchiectasis, in large abscesses and in tuberculous cavities, in which it refers abundant morning expectoration caused by the change in the patient' s position when getting up and cleaning the bronchus.

The viscosity or consistency of sputum depends on the amount of water, mucus and debris it contains, sometimes being very adherent, as in pneumonia and bronchial asthma; in others with a large necrotic component (purulent sputum), such as grangrena and lung abscess; or being extraordinarily fluid, as observed in acute lung edema.

The color and transparency depend on the amount of hemoglobin or bile pigments, foreign substances and the solution material: serosity, mucus, exudates. albuminous, etc. Thus, we can observe, in the direct examination of the sputum, the variants caused by dilutions of hemoglobin, that is: rusty, currant jelly, vinous, dark red or chocolate, all of them the product of conditions as different as pneumonia, pulmonary tuberculosis, bronchial carcinoma or amoebic liver abscess, which reflects that the existing lesion has affected blood vessels causing passive vascular congestion capable of giving rise to blood extravasations to small hemorrhages. Even more common is to observe green or greenish-yellow sputum resulting from bacterial suppurations by cocci or tuberculous bacteria.

Sometimes the discovery of blackish expectoration forces us to think about pulmonary anthracosis. All of the above abounds in the sense of the value that should be given to the color of sputum.

The odor, although not of great importance, sometimes has diagnostic value, as occurs in some pulmonary mycoses in which a characteristic yeasty odor is observed. Also in suppurative bronchiectasis or in putrid or gangrenous abscesses, extraordinarily fetid odors can be detected from the vomitus that these patients present, which ensure the presence of anaerobic germs.

The taste is usually not significant except when the patient reports a taste of blood, a symptom that often precedes the appearance of hemoptoic sputum; other times it indicates a salty taste in hydatid cyst and something bland, nauseating, in cavitary tuberculosis. Some diabetics report noticing a sugary taste in their expectoration during hyperglycemic crises.

The types of expectoration are grouped as follows:

- Mucosa.
- Mucopurulent.
- Purulent.
-Serosa.
- Bloody.

Mucous membrane. Transparent, colorless appearance with numerous air bubbles; It is usually observed in catarrhal bronchitis and bronchial asthma.

Mucopurulent. Consisting of accumulations of mucopus, which are individualized in the form of homogeneous masses in the spittoons, which is why they are also called "numular sputum"; They are yellowish white or slightly greenish in color, common in the final stages of catarrhal bronchitis, in secondarily infected bronchial dilations and in pulmonary tuberculosis.

Purulent. Greenish yellow coloration, opaque, more or less liquid, odorless or very foul; reflects a process

highly suppurative, which is evacuated through the bronchus to the outside, as occurs in lung abscesses or large bronchiectasis.

Serous. It is transparent, practically liquid, whitish or slightly tinged with pink and covered with abundant foam, has no odor, is almost always accompanied by dyspnea and is almost pathognomonic of acute pulmonary edema.

Bloody. It can range from bloody sputum to downright bloody expectoration. It is very common in pulmonary infarction, tuberculosis and bronchial carcinoma.

EMOPTYSIS

Concept

It is the expulsion by expectoration of the blood contained in the respiratory tract.

The blood in hemoptysis comes from the respiratory system. Although in exceptional cases it may be due to the rupture of an aortic aneurysm in the left trachea or bronchus.

When blood is swallowed, it can be vomited later, simulating hematemesis.

Semiognesis or pathophysiology

1. Pulmonary hyperemia:

a) Active inflammatory congestion.

b) Passive congestion.

2. Rupture of blood vessels:

a) Bronchial or pulmonary arteries.

b) Rasmussen aneurysms.

3. Arterial hypertension of the greater or lesser circulation.

4. Blood dyscrasias with coagulation disorders.

5. Aortic aneurysms.

In any of these mechanisms, the spilled blood awakens the cough reflex in the trachea.

Obstruction of the bronchi by blood causes cyanosis, dyspnea, cough, which sometimes accompanies hemoptysis.

semiography

Depending on its quantity, hemoptysis can be:

1. Small: sputum stained or streaked with blood expelled by cough.
2. Median: 100-120 mL of blood (almost always in tuberculosis).
3. Large: bright, withering blood, mouth full, with suffocation and rapid death. Sometimes the bleeding is not seen, due to fatal syncope, as occurs when a Rasmussen aneurysm ruptures due to ulceration of a large vessel, in tuberculosis.

The onset of hemoptysis can be sudden or preceded by retrosternal pain and flashes of color that are accompanied by tickling in the trachea, coughing and a special taste in the mouth.

As for quality, the blood is red, sparkling, airy; After the first expulsion has passed, it can be repeated when the patient becomes agitated and speaks.

After the hemoptysis, in the following days, the expectoration is dark, in clots in the form of bronchial molds. If red blood reappears at this stage, this is due to new bleeding.

The symptoms associated with hemoptysis are: tachycardia and fever, the latter when it intensifies is a sign of severity, almost always due to tuberculous generalization.

During the physical examination of the patient with hemoptysis, he should not move or be percussed; Only auscultation will be performed.

Auscultation gives us the signs of the injury that caused the bleeding or subcrackling rales due to blood in the bronchi.

The appearance of rales in a single lung allows us to assess the location of the causative process to apply the appropriate therapy.

Radiology sometimes makes it possible to exactly locate the bleeding lesion.

Positive and differential diagnoses

Hemoptysis can be confused with:

1. Epistaxis.
2. Stomatorrhagia.
3. Varicose veins of the rhinopharynx, oropharynx and base of the tongue.

wow

In these three cases the diagnosis is easily established by examining the bleeding region or the clot left by the bleeding vessel. In addition, the signs of hemoptysis and the pleuropulmonary signs of the disease that caused the hemoptysis are missing.

4. Hematemesis or vomiting of blood. It is preceded by digestive symptoms, is expelled with vomiting, is not aerated, has an acid reaction, with food residues and is followed by melena in subsequent days.

In hemoptysis, respiratory symptoms precede:

There is always cough and the blood is red, foamy and airy, with a neutral or alkaline reaction, without food remains and followed in subsequent days by hemoptoic expectoration.

Semidiagnosis

- Respiratory conditions.
- Circulatory conditions.
- General conditions or hemorrhagic syndromes.

Respiratory conditions

Pulmonary tuberculosis: evolutionary forms, non-evolutionary forms, cavitary forms.

1. Evolutionary forms. It can be: a symptom of the beginning of the disease or an evolutionary outbreak; In this case there are symptoms of tuberculous impregnation such as fever, tachycardia, general malaise, weight loss and sweating. It appears in pneumonic lesions or in caseation processes that ulcerate a blood vessel.
2. Non-evolutionary forms. Fibrous lesions with light, repeated bleeding, without fever, without tuberculous activity. Bleeding is due to vascular dilations and neoformations that occur in tuberculous fibrosis.
3. Cavitary forms. Profuse hemoptysis. It can lead to rapid death due to anemia, shock or asphyxiation.
   It appears in tuberculosis excavated by the rupture of a Rasmussen aneurysm due to an injury to the cavern wall.

It is also observed in the dilation of the bronchi or bronchiectasis, especially dry bronchiectasis, lung cancer (sputum in currant jelly); pulmonary infarction; mycoses; lung abscesses and gangrene; and pneumonia.

Circulatory conditions

They cause hemoptysis due to hypertension and stasis in the minor circulation:

1. Mitral injury: narrowing.
2. Aortic injury: aneurysm.
3. High blood pressure.
4. Congestive heart failure.

General conditions or hemorrhagic syndromes

1. Thrombocytopenia.
2. Acute leukemia.
4. Hemophilia.

VOMIC

Vomicing is the sudden evacuation to the outside, through the respiratory tract, accompanied by coughing and suffocation, of a generally purulent liquid collection that has just opened in the bronchi.

Semiography

Vomica may appear suddenly or preceded by bloody expectoration.

Clinically, pain is established, which may be absent, with the onset of coughing and the appearance of waves of pus that increase the coughing, accompanied by nausea, anxiety, intense dyspnea, cyanosis, sometimes leading to bilateral flooding of the bronchial tree that causes sudden death.

When the process progresses favorably, the pus expelled in successive bouts of coughing decreases, the patient is relieved, and dyspnea and cyanosis tend to disappear.

Sometimes the pus is expelled in small and repeated amounts, called fractionated omics.

Some authors accept that the pus that is eliminated from a tuberculous cavern or bronchial dilation causes the so-called pseudovomia.

Semidiagnosis

Its origin can be:

- Bronchopulmonary.
- Pleural.
- Mediastinal.
- Extrathoracic or abdominal.

Bronchopulmonary. Evacuation through a bronchus of the contents of a pulmonary or bronchial cavity. The material may or may not be purulent, as in:

- Lung abscesses.
-Giant tuberculous caverns.
- Infected saccular bronchiectasis.
-Suppurative lung cysts.
- Hydatid cysts (liquid like rock crystal).

Pleural. Evacuation through a bronchus of cystic pleurisy, almost always purulent, which may be due to:

- Pneumococcal pleurisy.
- Streptococcal purulent pleurisy.
  Tuberculous purulent pleurisy.

Mediastinal. Rupture in a bronchus, from a mediastinal abscess, determined by:

- Acute mediastinitis.
- Cold abscess of the mediastinum (TB).

Extrathoracic or abdominal. The purulent contents of the abdomen are evacuated through the diaphragm; it requires that the basal pleura be inflamed; The pus makes its way to a bronchus, the most common of which are:

a) Large liver abscesses. Amoebic: typical chocolate pus.
b) Perinephritic abscesses.
c) Subphrenic abscesses consecutive to:

Perforated gastroduodenal ulcers. Pericolecystitis.

RESPIRATORY SYSTEM. ALTERATIONS IN THE PHYSICAL EXAMINATION

34

GENERAL PHYSICAL EXAM

In this first section we will point out those data obtained in the general physical examination, which are very useful in respiratory conditions.

In the study of conditions of the respiratory system, the discovery of certain remote or distant signs has a fundamental value, which guides towards the diagnosis of said conditions. Among these signs we will study certain ocular alterations; To understand them better we will do a brief anatomofunctional reminder of ocular sympathetic innervation.

The pupil dilates (mydriasis) by the action of the iris dilator muscle, whose innervation depends on the sympathetic, and contracts by the action of the iris constrictor muscle, with parasympathetic innervation.

The center of sympathetic innervation is located in the spinal cord, especially at the level of segments D1, D2 or D3, although it extends throughout the cervical spinal cord to the medulla (ciliospinal center of Budge); The preganglionic fibers leave with the dorsal roots D1, D2 and D3 and go to the stellate ganglion (last cervical sympathetic ganglion joined to the first dorsal one), continue along the cervical sympathetic chain without stopping at the level of the middle cervical ganglion and reach the superior cervical ganglion where they synapse. From here the postganglionic fibers that surround the carotid originate, and with it they end up in the cranial cavity.

A part of these fibers reaches the ophthalmic ganglion and, without synapsing, joins the short ciliary nerves. The other does not go to the ophthalmic ganglion, but reaches the eye directly and forms the long ciliary nerves, which reach the iris dilator.

The parasympathetic innervation has: a preganglionic part that depends on the third pair and goes from the nucleus of this nerve in the mesocephalon to the ophthalmic ganglion, and another postganglionic part, which goes with the short ciliary nerves, from the ophthalmic ganglion to the constrictor muscle of the iris.

CLAUDE BERNARD ORNER SYNDROME

Semiognesis or physiopathology

This syndrome is observed whenever the sympathetic pathway of the eye is destroyed or inhibited by a pathological process that involves it in any part of its path.

The most frequent entities that affect this nerve are the processes of the apex of the lung that invade the sympathetic chain, especially the stellate ganglion.

As the excitation of the sympathetic produces mydriasis (due to its action on the iris dilator muscle), its destruction or inhibition will produce miosis. In this case, the parasympathetic action that acts on the iris constrictor muscle predominates.

The sympathetic innervates the Müllerian muscle, which helps elevate the upper eyelid; When this nerve is injured, the palpebral slit is reduced. Finally, Horner's muscle (which helps project the eyeball forward) is left without innervation in these cases, which explains the enophthalmia observed in sympathetic destruction.

Semiography

It is characterized by:

1. Miosis.
2. Enophthalmia.
3. Narrowing of the palpebral fissure.

Semidiagnosis

The diseases or processes that most produce this syndrome are pleuropulmonary lesions, whether infectious or tumorous, as long as they are located in the pulmonary apex and in the pleural dome.

POURFOUR DU PETIT SYNDROME

Semiognesis or pathophysiology

It is the opposite of that described in the previous syndrome, since sympathetic excitation occurs due to nervous irritation.

semiography

It is characterized by:

1. Mydriasis.
2. Exophthalmia.
3. Widening of the palpebral fissure.

Semidiagnosis

Same as the previous syndrome, but in the initial periods of these processes when they act by irritating the sympathetic.

ANISOCORY

It is the inequality of the pupillary diameters, but the reflection to light and accommodation is preserved.

Semiognesis or pathophysiology

It is due to the irritation exerted by the lesions of the pleuropulmonary dome on the third cervical sympathetic ganglion; It affects only the pupillodilatory fillets.

When the action is irritative, mydriasis occurs and if it is paralytic, miosis.

Semidiagnosis

1. Vertex pleuritis of tuberculous etiology; In this case, if the lesion is recent, mydriasis occurs and if the lesion is an old scar process, miosis occurs.

2. Cancer of the vertex. It behaves the same as vertex pleuritis.

Test for incipient anisocoria

In these cases the cause exists, but the anisocoria is latent. When dilating eye drops are instilled in equal doses in both conjunctival fornices, mydriasis is greater on the diseased side. This is the Contonet test, which is performed with atropine or cocaine.

IPOCRTIC FINGERS

Known by the names of hippocratic fingers, drumstick or drumstick; It was Trousseau who called them "club fingers" in a tuberculous patient (Fig. 34.1).

Semiognesis or pathophysiology

Clubbing fingers are due to soft tissue hyperplasia without bone alteration.

The fundamental mechanism appears to be tissue anoxia.

Fig. 34.1 Clubbing fingers.

Semiography

1. It consists of a special deformity of the last phalanx of the hands and feet, especially the hands.
   The phalanx becomes globular like the clapper of a bell.

2. The nail is curved like a spherical cap in watch glass. Sometimes the edge curves into a parrot's beak.

Semidiagnosis

1. Chronic bronchopulmonary diseases.
2. Chronic heart diseases, especially congenital heart disease with cyanosis.

IPERTROPHYING NEUMIC OSTEOARTHROPATHY

Semiognesis or pathophysiology

1. Tuft-shaped thickening of the phalanges.
2. Subperiosteal bone neoformation with rarefaction in the phalanges and metacarpals.

semiography

1. It presents the same characteristics as those indicated in the Hippocratic fingers, also observing what it con-
   We then point out.
2. Thickening of the carpal, carpometacarpal and more rarely tibiotarsal, elbow and knee joints.

3. Pain in the affected joints.

Semidiagnosis

Diseases in which both deformities appear can be (fig. 34.2):

Pulmonary

1. Chronic bronchitis and bronchiectasis.
2. Pulmonary emphysema.
3. Chronic pulmonary suppuration.

Fig. 34.2 Semidiagnosis of digital clubbing.

ATB RC LOS S I hippocratism
digitalis is common, but hypertrophic
osteoarthropathy

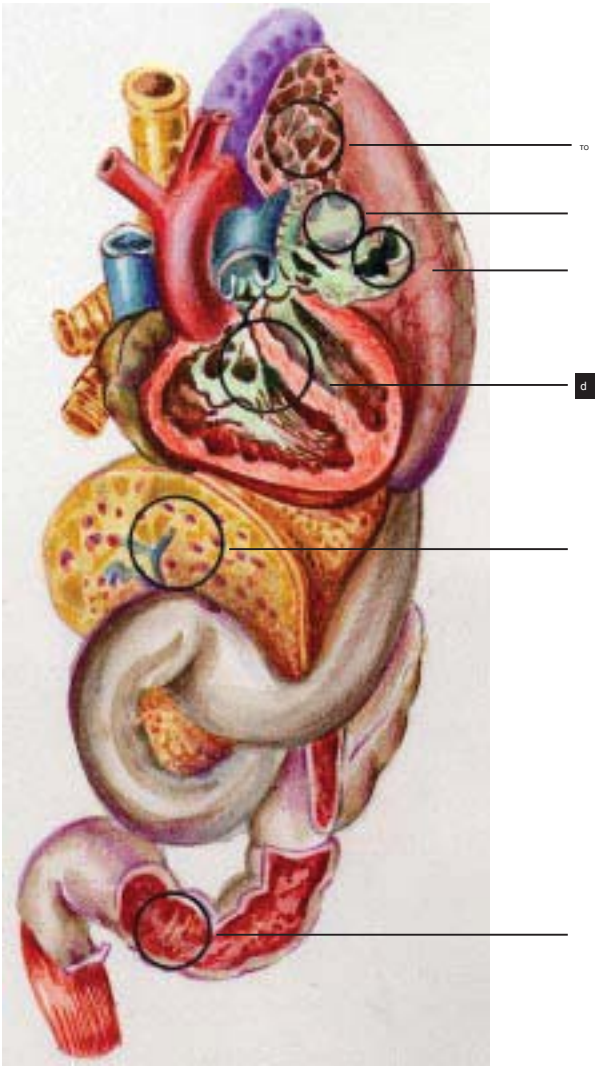
BT MOR SP LMONAR S The fingers
drumstick can be presented
several months before pulmonary
manifestations

C ABSC SO P LMONAR Through
the appropriate etiological therapy
digital hippocratism can disappear,
to recur with exacerbation
or recurrence of the primary condition

DT TRALOG A D FALLOT Congenital heart
conditions with ventricular septal defect
are invariably accompanied by cyanosis and
clubbing of the fingers.

C RROS SH P T CA Hypertrophic
osteoarthropathy occurs frequently in biliary
cirrhosis, but is rare in portal or post-necrotic
cirrhosis.

F COL T S LC RAT VA T digital
hypocratism is observed in gastro-intestinal
conditions accompanied by chronic diarrhea



4. Fibrous tuberculosis.

5. Lung cancer.

Circulatory

1. Cyanotic congenital heart disease.

2. Chronic heart failure.

Other exceptional conditions

1. Liver cirrhosis.

2. Long-lasting sprue, etc.

CHEST INSPECTION

The condition of the skin must be taken into account: color, scars, fistulous tracts, skin rashes, etc.; the state of the soft tissues: weight loss, obesity, collateral circulation, edema, tumors, pulsating empyema, muscle atrophy, etc.; and the configuration that constitutes the most important aspect and for its better study we classify it into:

-Normal tőthorax type (already described in the “Introduction to the Clinic” Section).

- Types of pathological chest.

-Unilateral thoracic deformities.

- Localized or circumscribed thoracic deformities.

TYPES OF PATHOLOGICAL CHEST

Tuberculosis or paralytic chest

Synonymy

Also called flat chest or permanent expiration (fig. 34.3).

Semiogenesis or pathophysiology

All alterations of this tőrax are due to marked thoracic muscle atrophies.

semiography

diameters. Anteroposterior: reduced; vertical; increased.

Bones. Clavicles: protruding (bull horn). Ribs: visible and oblique. Sternum: nothing of importance. Column: nothing of importance. Scapulae: winged.

Soft parts. Clavicular fossae: sunken. Intercostal spaces: sunken. Epigastric angle: acute. Shoulders: narrow, hence the deformed thorax appears.
Neck: long.

Semidiagnosis

Pulmonary tuberculosis and diseases that lead the patient to cachexia.

Chest emphysematosSO

Synonymy

Chest in a barrel or in permanent inspiration (fig. 34.4).

semiography

diameters. All increased. The anteroposterior may be larger than the transverse. This thorax is cylindrical and globular, and according to Laennec it is circular or hexagonal in cross section.

Bones. Clavicles: horizontal and elevated. Sternum: forward at the level of the ribs. Column: nothing. Scapula: nothing. Ribs: horizontal.

Fig. 34.3 Tuberculosis or paralytic chest: a, dorsal; b, profile; c, horizontal section (normal chest in dotted line).

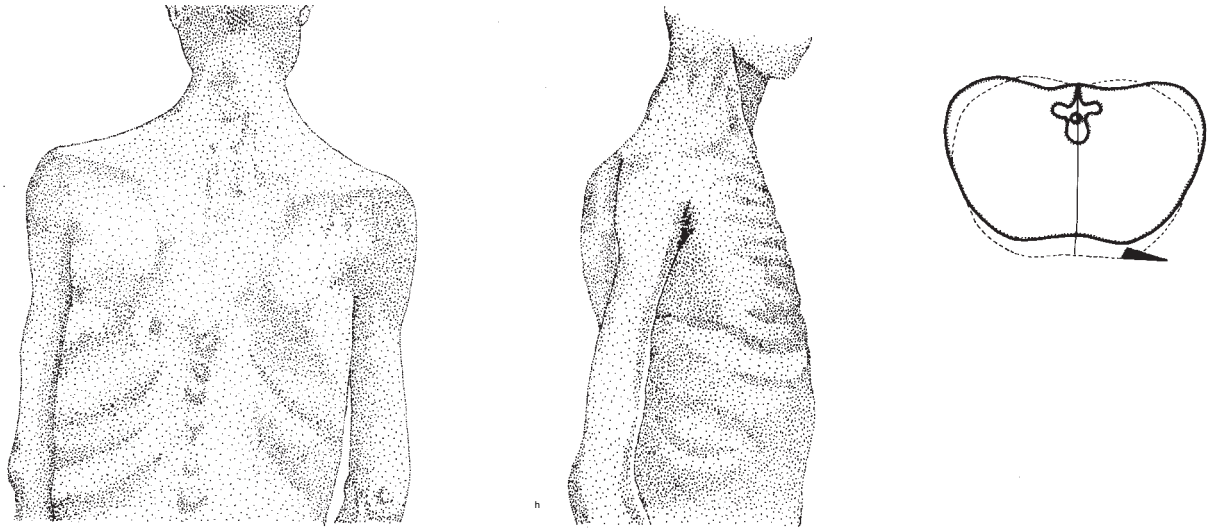


Fig. 34.4 Emphysematous chest.

Soft parts. Erased or bulging supraclavicular and infraclavicular depressions, very marked infraclavicular or cleidomamelonar protrusion of Louis. Intercostal spaces: widened and bulging. Epigastric angle: obtuse.

Semidiagnosis

Pulmonary emphysema.

Radial thorax

Synonymy

Thorax or keeled breast, or pectus gallinaceum (hen).

Semiography

Diameter. Transversely compressed; the anteroposterior diameter is elongated, and the transverse diameter is narrowed.

Bones. Clavicles: unimportant. Ribs: knots in the sternocostal joints that cause the so-called costal or ragquitic rosary. Sternum: projected forward and with a depression in the central region. This forward sternum causes the so-called keeled breast, also known as chicken or pigeon breast. Ribs: there is a lateral depression between the mammillary line and the fourth and seventh ribs. This transverse depression from the base of the xiphoid process up to the infraaxillary region originates the so-called Harrison's Groove. Scapula: nothing in particular. Column: nothing in particular.

Soft parts. Clavicular fossae: unimportant. Intercostal spaces: unimportant. Epigastric angle: unimportant.

Semidiagnosis

1. Rickets.
2. Obstacle in the lower airways in the child.
3. Whooping cough.

Infundibuliform thorax

Depression like the concavity of a funnel in the anterior plane, which goes from the middle of the body of the sternum to the xiphoid appendage, sometimes ascending to the third rib.

Synonymy

Funnel chest, pectus excavatus.

Semidiagnosis

1. Congenital deformity.
2. Rickets.

Cobbler's chest

Variety of infundibuliform thorax, which consists of a circumscribed depression in the xiphoid process.

Causes. It is produced by supporting hard bodies against the sternum, as occurs in shoemakers and carpenters.

Kyphoscoliotic chest

Defect in the direction of the spine that causes a characteristic thoracic deformity.

This type (kyphoscoliotic) is the combination of two bone malformations: a combination of kyphosis or hump in the upper part of the back with scoliosis or lateral alteration of the spinous processes of the spine.

This deformity is accompanied by one shoulder being higher than the other.

As the spine is twisted on its axis, the hemithorax that corresponds to the side of the cavity projects forward and the one that corresponds to the convexity projects backwards.

This thorax is accompanied by alterations of the intrathoracic organs and can lead to heart failure.

Semidiagnosis

1. Constitutional.
2. Vicious attitude in schoolchildren.
3. Rickets.
4. Pott's disease.

Conoid or enlarged thorax

It is due to abdominal diseases that increase the upper portion of the belly, such as: large ascites and hepatomegaly and splenomegaly.

This thorax has the shape of a cone with a base downward and apex upward.

UNILATERAL TORTIC DEFORMITIES

They are of two types:

1. Hemithoracic dilation.

2. Hemithoracic retraction.

Emithoracic dilation

semiography

Increase in the hemicircumference of that hemithorax.

Increase in anteroposterior diameter. Widened intercostal spaces. Elevated shoulder. Curved vertebral column with convexity towards dilation.

Semidiagnosis

It can be produced by:

1. Distension of the pleural cavity:

a) Pleurisies: serofibrinous, hemorrhagic, purulent.

b) Pneumothorax.

c) Pionneumothorax, hydropneumothorax, hemopneumothorax.

d) Pleural tumors.

2. Increase in lung parenchyma:

a) Lung tumors.

b) Massive pneumonia.

c) Unilateral emphysema.

Emithoracic retraction

Semiography

Narrowed diameters. Narrowed intercostal spaces, sometimes overlapping the ribs like roof tiles. Shoulder descent. Curved spine with concavity facing retraction.

Deviation of the mamelon towards the midline.

Semidiagnosis

Fundamentally, it can be due to two causes:

1. Pleural:

Extensive and total pleural symphysis due to fibrinous pleurisy not punctured in a timely manner or due to sequelae of purulent pleurisy.

2. Pulmonary:

a) Pulmonary atelectasis due to bronchial obstruction of inflammatory or neoplastic origin.

b) Tuberculous fibrothorax.

LOCAL TORICAL DEFORMITIES

0 CIRCUMSCRIBED

Localized thoracic deformities can be of two types:

1. Vaulting.

2. Depressions or retractions.

Vaulting

They can be produced by:

1. Partial emphysema, especially supraclavicular or infraclavicular.

2. Lung tumors.

3. Cystic pleurisies.

4. Pleural tumors.

5. Empyema of necessity.

Depressions or retractions

The most frequent causes are:

1. Limited pleural symphyses, consecutive to pleurisies of varied etiology.
2. Pleural fibrosis, especially tuberculous, where retraction is accentuated due to atrophy of the muscles of the chest wall.

3. Lung tumors that cause partial atelectasis.

SIGN OF THE STRING OR PLUMBLINE OF PITRES

Semiotechnics

The Pitres maneuver consists of stretching a thread or string, with a weight or plumb line at the end, from the middle part of the sternal fork to the pubic symphysis.

The separation of the xiphoid process from the thread is what is called the sign of the string or the Pitres plumb line. This separation becomes greater the more pronounced the deviation of the sternum.

To make it more evident, a dermographic pencil can be used. With this, a straight line is drawn that starts from the center of the sternal fork and ends at the base of the xiphoid appendage. Next, and from the same middle of the sternal fork, the string is taken with the plumb line to the center of the pubic symphysis. Normally, the line made with the pencil coincides with the path of the string.

When there is a pleural effusion, it can be seen that the two lines separate, due to the deviation of the sternum towards the side of the effusion. This separation can be measured and,

According to Pitres, it makes it possible to calculate the magnitude of the spill. He states that a deviation between 0.5 and 1 cm corresponds to spills of 1-3 L and that between 1 and 3 cm corresponds to spills of more than 3 L.

ALTERATIONS OF RESPIRATORY MOVEMENTS

When we study normal respiratory movements in Section I, we consider four fundamental aspects: respiratory type, frequency, rhythm and amplitude or thoracic expansion. His alterations are also based on those considerations.

Respiratory type

Respiratory alterations can be due to: exaggeration and investment.

exaggeration

1. In women it is generally due to a disturbance of the movements of the diaphragm, as seen in:
   a) Diaphragmatic pleurisy.
   b) Phrenic neuralgia.
   c) Diaphragmatic paralysis.
   d) Abdominal distension due to: meteorism, ascites, tumors and pregnancy.
2. In men it may be due to difficulty expanding the upper portion of the chest:
   a) Side tip of pneumonia and vertex pleuritis.
   b) Intercostal neuralgia.
   c) Rib fracture, etc.

investment

1. In women, it occurs against any process that makes upper rib mobility difficult.
2. In men the opposite: it occurs in the face of any process that hinders diaphragmatic mobility.

Frequency

The alterations have already been studied in the previous chapters, they can be due to:

1. Increase or polypnea.
2. Decrease or bradypnea.

Rhythm

It corresponds to the already known alterations of inspiratory and expiratory dyspnea, and arrhythmias of Cheyne-Stokes, Biot, Kussmaul, etc.

Thoracic breadth or expansion

Amplitude may be decreased in a hemithorax by:

1. Defense against pain, as occurs in pneumonia, pleuritis, etc.
2. Pleurisy with effusion.
3. Pneumothorax, etc.

PALPATION OF THE CHEST

This examination complements the data obtained by the inspection, adding details such as alterations in thoracic sensitivity and elasticity and in the intensity of vocal vibrations or thrills.

CONDITION OF THE SKIN AND SOFT PARTS ON PALPATION

Fur:

In it we will observe the following alterations: disappearance of cyanosis due to compression, which does not occur in other colorations; peeling of pityriasis: presence of scars, fistulas, rashes.

Soft parts:

1. Exploration of the subcutaneous cellular tissue and muscles where we will check for myoedema or idiomuscular contraction in pathological cases.
2. Weight loss, which can appear in chronic lung conditions, especially tuberculosis.
3. Collateral circulation; In it we can study emptying and refilling by palpation and thus know the direction of blood flow.
4. Edema; Above all, it allows you to appreciate the saucepan or godet.
5. Subcutaneous emphysema; Palpation allows the characteristic crepitation to be evident. As we know, this emphysema appears in mediastinal wounds, spontaneous pneumothorax and gas gangrene of the chest wall.
6. Start of need; Palpation allows us to verify not only the fluctuation of the pus, but also the disappearance of the tumor upon compression with the fingers.
   A variant of this empyema is pulsatile empyema, which can be confused with an aneurysm, especially of the aorta if we are in the thoracic region, but palpation reveals, when it is an aneurysm, the expansion of this vascular tumor.
7. Lymph nodes; Palpation not only locates them, but also studies their characteristics. This examination is important for the diagnosis of tuberculosis, cancer, etc.

   In particular, according to Sergent, supraclavicular adenitis and lymphangitis are diagnostic elements in tuberculous apical pleuritis.
   It is interesting to note that, sometimes, in the left supraclavicular region near the sternocleidomas-

lymphadenopathy appears without the other cervical lymph nodes being enlarged. This sign, described by Troissier by whose name it is known, has a high semiological value and almost always indicates the existence of an abdominal carcinoma.

8. Contractures; They are very important, according to Pottenger, since in the case of a visceromotor reflex, one can, depending on the contracted muscle group, locate the injured viscus. So, for example:

   Injuries to the upper lobe produce contracture of the muscle group formed by the trapezius; pectorals, scalenes and angular of the scapula.

   Pleurisies give rise to contracture of the muscles of the vertebral canals, causing the so-called Ramond's spinal sign.

   Malignant tumors produce contracture of the muscles of the chest, neck, face and abdomen on that side.

9. Atrophies; As we know, chronic pleuropulmonary processes, particularly tuberculosis, usually cause muscle atrophies in the affected region.

   Finally, palpation of the muscular plane allows us to verify myoedema in chronic pulmonary processes such as tuberculosis and cancer.

TOR CICA SENSITIVITY

It is important because it allows us to study the provoked pain by palpation, and to check the spontaneous pain, specifying its characteristics.

TOR CICA ELASTICITY

Semiognesis or physiopathology

Pathologically, elasticity can be: increased or decreased.

Increased

Unimportant, it appears rarely in rickets and osteomalacia.

diminished

It is due to alterations in the flexibility of the rib cage or physical variations in its contents.

The decrease can be:

Bilateral. As seen in emphysema, where there is a decrease in lung elasticity and cartilaginous ossification.

Unilateral. As it appears in pleurisy, tumors or large condensations.

Semidiagnosis

Pathological modifications of respiratory thoracic expansion can be:

Bilateral

These bilateral alterations of tőthorax expansion can be of two types:

Increased expansion. Exaggeration of the respiratory type

Decrease. Emphysema, tuberculous pulmonary sclerosis or not; obstruction of the upper airways; bilateral pleural effusions; painful disorders of the chest wall.

Unilateral

In this case the alterations are due to a decrease in expansion and the causes that determine them are:

intense chest pain, pleurisy with effusion, pneumothorax, symphysis, extensive pleurisy, pulmonary atelectasis, pulmonary sclerosis, tumors, etc. The expansion is increased in cases of respiratory replacement.

Located

They also occur due to processes that reduce respiratory expansion. At the vertex they constitute an early sign of pulmonary tuberculosis. At the base they are due to small spills.

FREMITO 0 VOCAL VIBRATIONS

Now knowing the origin and transmission of vocal vibrations or thrills, described in Section I, we can better understand the causes that determine their alterations in the different pathological processes.

Semiognesis or pathophysiology

1. In bronchial obstruction, vocal vibrations are not appreciated because the obstacle that closes the bronchus prevents the propagation of the sound wave to the corresponding lung territory.
2. In pneumothora, emphysema and pleural effusion, as there is a great variation in density between the media that the sound must pass through, it is diffused and dispersed, so vocal vibrations are greatly diminished or abolished.

3. In caseous pneumonia and tuberculosis, because there is a more homogeneous medium that favors transmission and prevents reflection, vocal vibrations are transmitted more easily and appear increased on palpation.

Remember that, as discussed in Section I, vocal vibrations “ run well, fly badly, and swim worse.”

PATHOLOGICAL MODIFICATIONS OF VOCAL VIBRATIONS

Semiography and semidiagnosis

Vocal vibrations can be found: increased, decreased, abolished and preserved.

Increased vocal vibrations

Vocal vibrations increase in intensity for three reasons:

1. Pulmonary hyperventilation. Functional substitution appears in healthy regions of the lung. Thus, for example, we find it at the level of the infraclavicular region accompanying the increase in intensity of the percussive sound or scudism and the puerile breathing of medium-caliber pleural effusions.
   Sometimes vocal vibrations spread to regions where they should be missing, as occurs on the surface of a pleural effusion or a pneumothorax, and are then called return or indirect vocal vibrations. Other times they are increased in an entire hemithorax when it replaces the function of the opposite hemithorax.

2. Lung condensations. In this case, for the vocal vibration to intensify it is necessary that the lesion be superficial, of sufficient volume and that the bronchus be patent.

   They appear in: inflammatory pneumonia, pseudolobar bronchopneumonia, tuberculous infiltration and with solid lung tumors, as long as the bronchus is not obstructed.

   The fundamental factor that determines the increase in intensity of vocal vibrations in condensations is the existence of a homogeneous medium and a patent bronchus.

3. Pulmonary caverns. For the caverns to determine an increase in vocal vibrations, they must be of good size (4 cm in diameter) , be superficial, and have a patent bronchus.

   Vibrations appear in: tuberculous caverns, lung abscesses and bronchiectasis.
   The caverns increase the intensity of vocal vibrations due to the better conductivity of the condensed pericavitary tissue and because the cavern acts as a resonance chamber.

Decreased vocal vibrations

They may be due to:

1. Phonation disorders. Inflammatory or ulcerative laryngitis, paralysis of the vocal cords.
2. Increased thickness of the chest wall. Edema. subcutaneous emphysema, obesity.
3. Respiratory processes. Those that prevent air conduction due to an obstacle in the bronchi or that increase sound reflection due to the presence of media of different density.

   Causes. Bronchial obstruction due to a foreign body; tumorous compressions of the bronchi; pulmonary emphysema; liquid and gaseous pleural collections such as: pleurisy with effusion, pneumothorax, hydropneumothorax.

Abolished vocal vibrations

They appear in the same previous processes when they act with maximum intensity.

This is what happens in:

1. Complete bronchial impermeability.
2. Massive pneumonia that fills the bronchi.
3. Large pleural effusions.
4. Pneumothorax.
5. Thick pleural symphysis or pachypleuritis.

Preserved vocal vibrations

Vocal vibrations are preserved in pathological processes that are not very extensive, or very deep, which, therefore, do not alter them:

1. Congestion and edema of the lung.
2. Small condensations.
3. Pleuropneumonia, in which the pneumonic factor increases them and the pleural reaction decreases them; therefore, there is no alteration.
4. Enguisted pleurisies (insufficient volume of the lesion).
5. Deep processes: pneumonia and deep caverns.

PLEURAL ROB

Concept and semiogenesis

It is due to the friction of the inflamed pleural sheets, which when normally sliding with respiratory movements do not produce any palpatory sensation, but in this case, being inflamed they give rise to a palpable vibration accompanied by noise.

semiography

The pleural rub is heard and palpated in the infrascapular and infraaxillary regions during both times of breathing, resembling the vibration of the crackling of snow or the flexing of new leather.

Semidiagnosis

Pleural rub appears in acute dry pleuritis and in pleurisy with effusion before its installation and after its disappearance.

PALPABLE RALES OR BRON UIIAL FREMITS Concept

and semiog nesis

Also called dry, hoarse or wheezing rales;

They are caused by the passage of the air column through the bronchi whose lumen is narrowed by the inflammation of its walls, or by the presence of an adherent viscous secretion. They are seen in both respiratory periods and, sometimes, disappear when secretions are mobilized by coughing.

Semidiagnosis

Bronchitis and asthma attacks.

PERCUSSION OF THE CHEST

PATHOLOGICAL MODIFICATIONS IN THE INTENSITY OF PULMONARY PERCUSSION NOISE

Semiognesis and semidiagnosis

Aume tada or iper or ority iperresonance

i Tympanism

i my uida or ipo or oridad

Subdullness

Dullness

Hypersonority

It is due to the increase in the air content of the lung with a decrease in density, or because percussion vibrates the air in pleuropulmonary cavities (caverns and pneumothorax). The great tension of the air transforms hypersonority into hyposonority.

Hypersonority can be of two types:

1. Hyperresonance.
2. Tympanism.

1. Hyperresonance. It occurs for two reasons, which are:

a) Substitution. The typical example is found in medium-caliber pleural effusions, in which the infraclavicular region becomes hyperresonant (a sign described by Skoda).

b) Emphysema. In this, the entire thorax is conceived as hyperresonant, especially the infraclavicular region, and the cardiac dullness disappears due to the presence of emphysematous lung flaps.

2. Tympanism. Appears in:

a) Pneumothorax. Where it has a low tone, except when the air is under high tension.

b) Cavities. Both in bronchiectasis and abscesses.

For a cavern to cause percussive tympanism, it must be superficial, more than 4 cm in diameter, with a patent bronchus and without any content inside.

Hyposonority

It is due to two main causes:

1. Lung condensations with increased density.
2. Interpositions of liquids, solids and gases at high tension.

In both cases the sound that originates will have lower intensity, higher pitch and shorter duration.

The hyposonority variants are:

1. Submattness. This appears in:

a) Pneumonia at the beginning.

b) Pleurisy with little effusion.

c) Upper limit of medium caliber spills.

2. Absolute dullness. This appears in:

a) Large condensations as in pneumonia.

b) Tuberculous caseous blocks.

c) Large effusions, causing the so-called water dullness, which is accompanied by an increase in resistance to the percussing finger (Tancuam percusso femoris: like percussing a thigh).

d) Pachypleuritis, in which cases the vibration of the aerated area of the lung is suppressed and the sound is produced by the percussed finger.

SPECIAL STUDY OF PERCUSSION OF PLEURAL EFFICIENCY

Medium volume pleurisies

The percussive characteristics of pleural effusion correspond not only to the absolute dullness that they usually give, but also to the loss of elasticity or increase in resistance that they oppose to the percussing finger.

These characters correspond to spills of medium volume, that is, more than half a liter, since small ones tend to give submattness to the percussion.

The upper limit of the spills is separated from the Absolute Loudness Zone by a subdullness line.

The upper limit is variable, according to the size of the spill: thus we have:

Small spills: horizontal line.

Medium spills: parabolic curve.

Large spills: horizontal line.

In medium volume spills we have pointed out that the upper limit of dullness describes a parabolic curve called Damoiseau parabola or Ellis line. This line ascends from inside to outside, originating behind

level of the spine, it crosses the shoulder blade and has its upper limit at the level of the armpit, where its maximum axis is located; then descends obliquely towards the sternum (fig. 34.5).

Garland's triangle is an acute angle formed between the vertebral column and the posterior branch of the parabola or Damoiseau curve (see Fig. 34.5). In this area, normal loudness can be replaced by a subdullness about 3 cm wide and this seems to be due to atelectasis of the lung next to the hilum.

Grocco's triangle. Di'ipofonesi zone for vertebrale opposta. It is a right triangle with a base or leg less than 2-8 cm lower limit corresponding to the base of the thorax (see Fig. 34.5). Greater leg following the line of the spinous processes of the corresponding vertebrae and hypotenuse prolonging the Damoiseau curve. In this triangle there is not only percussive dullness or subdullness, but also abolished vocal vibrations and absence of vesicular murmur.

It is considered that this triangle with these characters may be due to:

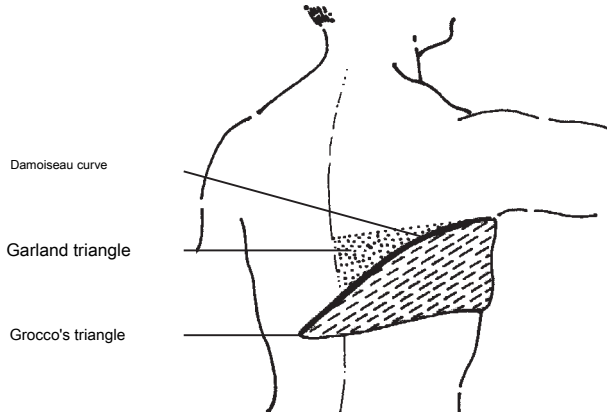
1. Small effusion in the opposite pleura.
2. Displacement of the mediastinum.
3. Disturbance of the vibratory capacity of the spinal column.
4. Distension of the interaorticoesophageal and interaorticoesophageal pleural fornices.

In medium-volume spills we find percussion above these depths, sonority in the posterior plane and true hyperresonance, sometimes tympanic, in the anterior plane, the latter called a scudism in honor of the author (Skoda) who discovered it.

Pleurisies in uistadas

They are liquid effusions that establish themselves in regions of the thoracic cavity isolated by pleural adhesions. They are almost always due to pneumopurulent pleurisy.

Fig. 34.5 Percussion of the chest in pleural effusion.



coccal or tuberculous serofibrinous or purulent pleurisies.

Cystic pleurisies can be:

- Interlobar.
- Mediastinal.
- Diaphragmatic.
- Mediastinum-diaphragmatic.
- From the great cavity.

Interlobar. They give the percussion a dullness along the path of the interlobular fissure, at the level of the axillary or dorsal region, calling it "sandwich dullness."

Mediastinal. If the fluid cysts in the anterior mediastinum, it will give a triangular dullness next to the lower portion of the sternum.

If it encysts in the posterior mediastinum, it will give a vertical band of dullness next to the spinal column.

Diaphragmatic. They give a horizontal band of dullness of the last two intercostal spaces, posterior and lateral, of the hemithorax. They are usually accompanied by pain due to phrenic neuralgia.

Mediastinum-diaphragmatic. They give a characteristic square of dullness, with a vertical mediastinal and horizontal band at right angles by association of the two previous ones.

From the great cavity. The dullness is variable in location. Sometimes, dull and tympanic percussion above when there is a hydropneumothorax.

AUSCULTATION OF THE CHEST

SEMIOTECHNICS OF AUSCULTATION OF THE RESPIRATORY

Auscultation is one of the most important, if not the most important, of the physical examination methods for the diagnosis of respiratory conditions.

In Section I we describe the essential requirements to perform optimal auscultation of the respiratory system. Here we will refer to the auscultation techniques for the bedridden patient. Remember that the thorax must be naked, at least in the part to be examined, always respecting modesty, and the auscultation must be done without the interposition of clothing.

If the patient must remain in bed, due to his illness or medical prescription, the anterior thoracic region will be initially auscultated, with the patient in a supine position and with a single pillow, to make auscultation of the supra- and infraclavicular regions easier. According to

the region to be explored, the person is asked to turn their head to the opposite side, not only for better exposure of the area being explored, but to avoid exhalation air on the examiner, with possible risk of contamination of communicable diseases in this way.

To examine the posterior region, the patient is sat on the bed with the cooperation of an assistant, who will support him while we do the procedure. Along the way, we listen to the lateral regions.

If the patient's prostration is very marked, if he or she is in a passive decubitus position, or if we do not have an assistant, the posterior region will be auscultated by placing the patient, first in left lateral decubitus and then in the right, to perform the auscultation of the right side and the left side, respectively. Another option, when the previous ones are impossible, is to slide the stethoscope receiver under the patient.

In Section I we also explain normal respiratory sounds, including the main elements of the vesicular murmur such as: intensity, tone, timbre, and rhythm.

After carefully specifying these details during auscultation, we must go on to identify adventitious noises, such as rales.

The examination will never be complete if the examined subject is not auscultated while speaking (natural and whispering voice) and afterwards, during the induced cough (to differentiate the unfolding rales, other adventitious sounds and lung murmurs, which had not been perceived in the previous auscultation).

One or two bursts of cough at the end of expiration are even recommended, as they are very useful to highlight these adventitious sounds and lung murmurs that had not been previously perceived.

The use of cough is very valuable in areas suspected of alteration or doubtful auscultation. With it, the so-called unfolding rattles disappear.

A very important factor is periodic auscultation of the patient. In this way, signs that had not been detected in previous examinations, because they did not manifest or had gone unnoticed, can be perceived. Also, others that had already been heard may disappear or be modified, which may have diagnostic and prognostic implications.

Finally, it is timely and important for the beginner to know that there are three requirements or laws, according to Ameuille, for lung conditions to have stethoacoustic translation and therefore allow an auscultatory diagnosis. They are the following:

1. Law of minimum volume: that the lesions have sufficient volume.
2. Law of minimum depth: that the lesions be sufficiently superficial.
3. Law of transmission: that the lesions are in an explorable region.

Not knowing, forgetting or dismissing these laws can fill us with perplexity and discouragement in some cases, which require the help of complementary tests, particularly imaging, for their diagnosis.

It is clear, then, that despite its enormous diagnostic value, auscultation of the respiratory system may have its limitations, but it cannot be replaced with imaging, which it must always precede.

PATHOLOGICAL MODIFICATIONS OF RESPIRATORY NOISES

Before beginning this topic, it is recommended that you review the normal respiratory sounds and their physiological modifications (glottic murmur, vesicular murmur, and bronchovesicular breathing) described in Section I, Chapter 9.

Semiognesis and semidiagnosis

- Pathological modifications of intensity.
- Pathological modifications of tone.
- Pathological modifications of the timbre.
- Pathological rhythm modifications.

Pathological intensity modifications

The intensity of the vesicular murmur can be of three types:

1. Strong breathing (exaggerated, childish or supplementary).
2. Weak breathing.
3. No breathing.

Strong breathing. In it there is an increase in intensity without modification of tone and timbre. It reflects the functional overactivity of the lung close to the diseased area or the functional substitution of one lung when the other is affected.

Appears in:

Condensations: in pneumonia, tuberculosis, tumors;
breathing is heard in neighboring unaffected areas.

Pleural effusions: in this case it is heard in the infraclavicular region accompanying the scudism and increased vibrations in medium-caliber effusions, or, on the other hand, in large effusions. When this breathing occurs with a harsh timbre, it is a sign of beginning condensation.

Weak breathing. In this case there is a decrease in intensity and its greatest importance is acquired when

It is one-sided. If it is bilateral, in order to give it value as a sign of a pleuropulmonary lesion, cases of poor mobility of the thorax or great thickness of the wall must be ruled out.

It appears in the form:

Bilateral: upper airway obstruction and pulmonary emphysema.

Located in a hemithorax: extrinsic or intrinsic obstruction of the main bronchus on that side due to tumors, mediastinitis and aortic aneurysms. Antalgic immobilization of a hemithorax. Unilateral emphysema.

Circumscribed: it is a sign of incipient pulmonary condensation and appears in: pneumonia, pulmonary tuberculosis, pleuropulmonary tumors and pleural

No breathing. It appears in: large effusions, extensive thick pleuropulmonary symphyses, total pneumothorax, bronchial obstruction (cancer) and massive pneumonia that fills the bronchus.

Pathological tone modifications

These join the timbre and intensity characterizing bronchovesicular respiration.

There are two tone alterations:

Low or severe breathing (very rare).

High or sharp breathing. In it the tone rises especially during expiration, transforming into blowing respiration.

The expiratory tone rises, reversing the normal relationship that exists between inspiration and expiration. This prolonged expiration acquires characteristics that bring it closer to the glottal breath.

Appears in:

Condensation of any type not large enough to transmit laryngotracheal noise (tubal murmur).

Tracheobronchial lymphadenopathy.

Mediastinal tumors.

In these latter cases, breathing becomes blown due to partial transmission of glottal vibrations.

Pathological modifications of the timbre

In pathological conditions the soft timbre of the vesicular murmur becomes harsh, grainy, dry.

Rough breathing. It is nothing more than a mixture of vesicular murmur and glottal murmur, that is, a pathological variant of bronchovesicular respiration.

Appears in:

Incipient pulmonary condensation.

Bronchitis, in this case, is temporary and mobile.

Pathological rhythm modifications

They are of three types:

1. Frequency modifications.
2. Modifications of the relative duration between inspiration and expiration.
3. Modifications in the continuity of the vesicular murmur.

Frequency modifications. This is altered for the following reasons:

Increased rate: tachypnea or polypnea.

Decreased rate: bradypnea.

Rhythm alterations: rhythmic arrhythmias, Cheyne-Stokes breathing, Biot, Kussmaul, etc.

Changes in the relative duration between inspiration and expiration. This alteration is carried out by lengthening the exhalation, which normally does not exist or is the third, fourth or fifth part of the inspiration. This causes the so-called prolonged expiration, which is basically blowing breathing or a pathological variant of breathing.

bronchovesicular tion.

Appears in:

Incipient condensation, almost always tuberculous.

Modifications of lung elasticity, as occurs in pulmonary emphysema where expiration is lengthened and the intensity of sound decreases, causing emphysematous breathing.

Modifications of the continuity of the vesicular murmur.

This may be interrupted by regularly spaced pauses, giving rise to the so-called Raciborski gasping.

It appears in the form:

Generalized: unimportant, since it is typical of nervous individuals or those with tremors.

Localized: in this case it has different origins:

Pulmonary origin, difficulty in inspiratory expansion of a lung area due to pleural adhesions as occurs in tuberculosis.

Pleural origin, due to pleural adhesions that prevent lung expansion (this is the most frequent and important cause).

Bronchial origin, partial obliteration of a group of bronchioles due to secretions and therefore disappears with cough.

Cardiac origin, the interruptions depend on the heartbeats that act on the pulmonary tongue.

LUNG RUMPS

Concept and semiog nesis

Pulmonary murmurs are nothing more than the glottic murmur transmitted by a pathological process beyond the thoracic territory where it should normally be heard and which therefore suffers the modifications of the lesion that causes it. They are caused by the passage of air through a duct or opening.

The glottal murmur is altered in the following way:

- If it undergoes small modifications, it constitutes a tubal murmur.
- If it acquires a hollow tone inside a cavity that acts as a resonance chamber, it is called a cavity murmur.

If it acquires a musical or metallic tone inside a large air cavity or a pneumothorax, it is called an amphoric murmur.

Tubal murmur (fig 34 6)

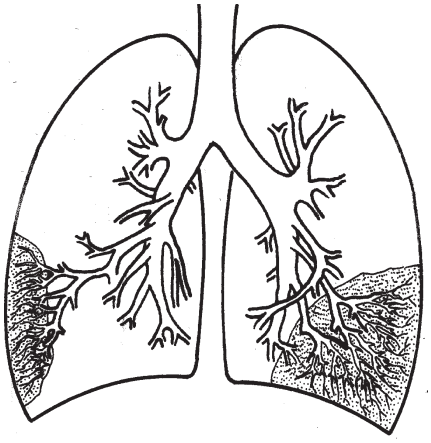
Synonymy. Bronchial respiration.

Semiogenesis or pathophysiology. Lung condensations that replace the normal lung parenchyma (poor conductor) with dense and uniform tissue (good conductor).

Necessary conditions:

1. Superficial or near-surface condensation.
2. Depth of the lesion that reaches the bronchi of 3 mm.
3. Sufficient volume of the lesion.
4. Good chest expansion.
5. Bronchial patency.

Fig. 34.6 Tubal murmur, diagram of two pulmonary condensations: a, superficial condensation that does not reach the bronchi of 3 mm in diameter; There is therefore no transmission of the bronchial murmur; b, deep condensation that does cause a tubal murmur.



Semiography. It is characterized by:

1. Intensity: variable, generally large, “it gets into the ear.”
2. Tone: high-pitched, no more than the glottal tone because the glottal murmur is transmitted through finer bronchi.
3. Timbre: harsh (similar to blowing through a hollow tube), but less harsh than the glottal blow.

It is imitated by quietly pronouncing the letters A, E, O.

Semidiagnosis. It is presented in:

1. Pneumonia.
2. Pseudolobar bronchopneumonia.
3. Pulmonary tuberculosis (caseous pneumonia).
4. Tracheobronchial lymphadenopathy.
5. Lung cancer (without bronchial obstruction).
6. Mediastinal tumors.

pleural murmur

It is a variant of the tubal murmur that appears when there is atelectatic condensation of the lung due to a pleural effusion.

Semiography. Soft, veiled, distant, expiratory, sharp as when pronouncing the letters E, I.

Semidiagnosis. Appears in:

1. Upper limit of medium spills.
2. The entire extent of sheet spills.

It is not observed in small or large spills.

Cavernous murmur (fig 34 7)

Synonymy. Cavitary murmur.

Semiogenesis or pathophysiology. Tubal murmur that modifies its timbre and tone in the presence of a smooth-walled superficial cavity 4 cm in diameter and with a patent bronchus. Pericavitary condensations favor the appearance of the murmur.

Semidiagnosis. Appears in:

1. Tuberculous caverns.
2. Lung abscesses (voids).
3. Bronchiectasis:

a) Large pleural effusions that compress the lung against the hilum. In this case, the thick bronchus acts as a cavity and the murmur is audible in the interscapulovertebral region.

b) Lung condensations that contact the trachea and bronchi.

c) Pleuropulmonary retractile fibrosis of the superior lobe rior that draws the trachea to that side and the murmur appears outside the sternum.

Fig. 34.7 Cavernous murmur.

Amphoric murmur (ig

34 8) Semiogenesis or pathophysiology. It is a tubal murmur modified by a superficial pulmonary cavity of more than 6 cm in diameter, with smooth, tense, empty walls and a patent bronchus. Or, modified by the cavity of a tension pneumothorax when it communicates with a bronchus.

Semiography. It has the following features:

1. Intensity: weak, although stronger on inspiration.
2. Tone: serious.
3. Doorbell: metallic.

It is imitated by blowing through the neck of a jug or amphora; sometimes it is heard making the patient cough.

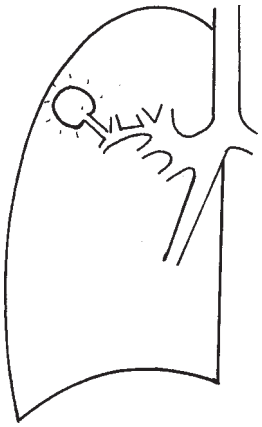
Semidiagnosis. It usually appears in:

1. Large caverns, especially tuberculous.
2. Pneumothorax: partial or total, where it appears when pleural pressure increases or when there is a pleuropulmonary fistula.

monar

3. Hydropneumothorax.

Fig. 34.8 Amphoric murmur.



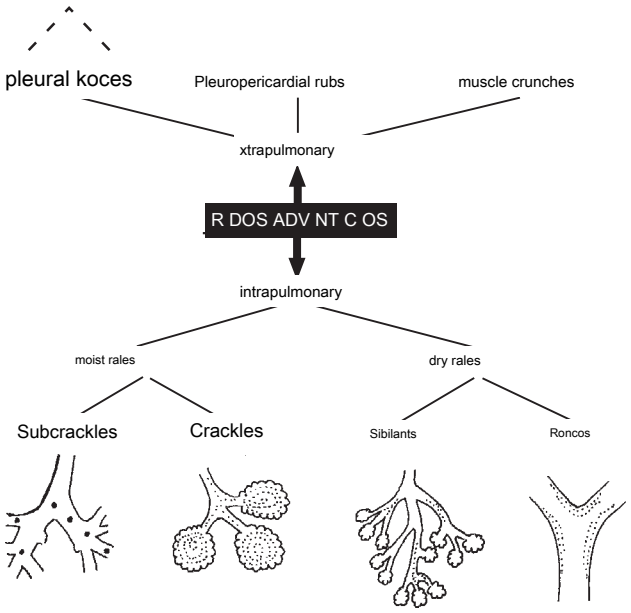


Fig. 34.9 Adventitious noises.

ADVENTICIOUS OR OVER-ADDED NOISES FIG.

Concept and semiog nesis

They are nothing more than added noises that occur in one or both phases of breathing.

In general, adventitious sounds are divided into extrapulmonary and intrapulmonary

Extrapulmonary sounds generally correspond to the transmission of noises caused by the sliding of the visceral pleura over the parietal pleura when both, or one of them, are affected by a pathological process; They can be:

1. Pleural rubs or friction.
2. Pleuropericardial rubs or rubbing (rare).
3. Muscle crunches.

The latter have nothing to do with the pleura and do not express any pathology, but they must be kept in mind to avoid diagnostic confusion.

Intrapulmonary sounds are generally produced by the passage of air over the secretions of the bronchial tree; They can be:

1. Wet rales: crackles or subcrackles.
2. Dry rales: hoarse and wheezing.

In all these adventitious noises there are several elements that we must study:

1. Stetoacoustic characters.
2. Place where noises are heard and mobility or fixity.
3. Breathing time in which they are perceived.
4. Sensations that supply palpation.

5. Modifications suffered by pressure, breathing, coughing and expectoration.

Extrapulmonary adventitious sounds

Pleural friction or friction. Two specific types can occur:

I. Large and dry chafing.

II. Discreet and moist rubs.

Large, dry rubs

semiography

1. They are intense noises that resemble:

a) Rub new korea.

b) Noise of the parchment or sheet of tissue paper being crushed between the fingers.

c) Noise of crushed ice being compressed with the hands. They are perceived as irregularly spaced, as if sliding the fingers over the dorsum of the metacarpophalangeal joints, with the palm of the hand on the pinna of the ear.

2. They appear in various regions of the thorax, but preferably on the sides; They are fixed and audible in the same place; They disappear when the spill occurs.

3. They are heard in both breath times.

4. They provide a special vibration called palpable pleural friction or pleural thrill.

5. They increase with pressure; They are not changed by coughing, breathing or expectoration.

6. Your differential diagnosis must be done with:

dry, hoarse and wheezing rales; These are less superficial, continuous, variable, mobile; They do not increase with pressure and disappear with breathing, coughing and expectoration.

Semiogenesis or pathophysiology

Its production mechanism is due to the dulling or thickening of the pleurae, which produce a harsh friction when they are inflamed and covered with fibrin.
These noises disappear during the spill phase, hence they are called indux and redu, that is, before the appearance of the liquid or after its disappearance.

Semidiagnosis

It is observed in:

1. Dry pleuritis. Almost always of tuberculous etiology. In it, sometimes the rubbing is very discreet and is called pleural rugae.

2. Pleurisy with effusion. In these cases, pleural friction friction can always be easily detected in the phase prior to the appearance of fluid. Once collected the effusion, they are only distributed above its upper limit, reappearing again throughout the entire height of the affected pleura when the liquid disappears; In these cases they are called pleural friction rubs.

return.

Discreet and moist rubs

Also called friction-rales, they are considered by some to be rales originating in the cortical region of the lung. They are heard at the end of inspiration and at the beginning of expiration. They occur superficially. They are fixed and cannot be modified by cough and breathing, hence their differential nature with subcrepitant rales. They appear in corticopleuritis.

intrapulmonary adventitious sounds

Rattles. "Any unnatural noise" produced by the passage of air during breathing, when passing through liquids found inside the lumen of the bronchi, or when this air passes through a narrowed duct, will be a rattle.

There are two types of rales:

I. Dry, vibrating or audible rales:

a) Roncos (snoring).

b) Sibilants.

II. Wet, mucous, or bubbly rales:

a) Crackles.

b) Subcrackles.

c) Cavernous.

Dry, vibrating or audible rales A. Hoarse rales (snoring).

First variety of dry rales; They are made up of high-intensity low-pitched adventitious noises, which are perceived by the patient himself and from a distance.

Semiogenesis or pathophysiology (fig. 34.10)

Hoarse rales are caused by the passage of air through the narrowing of the lumen of the medium and large caliber bronchi by:

a) Inflammatory swelling of the mucosa.

b) Viscous and adherent secretions.

c) Extrinsic bronchial compressions.

d) Intrinsic bronchial obstruction.

It can be said that cornage is a variety of snoring caused by the same process in the thick bronchi and trachea.

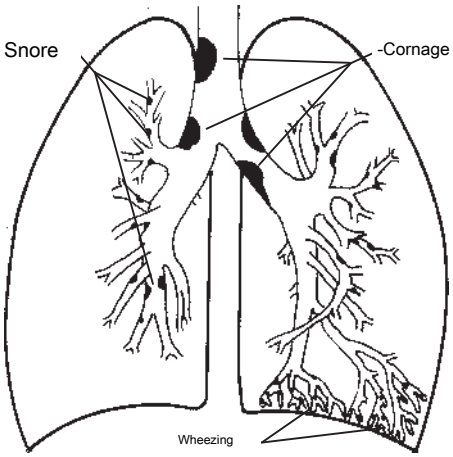


Fig. 34.10 Hoarse and wheezing rales.

semiography

1. They resemble:

a) The snoring of a sleeping man.

b) The note of a double bass.

They give the sensation that they arise deep in the thorax, hence their difference from friction.

2. They are heard in all regions of the thorax; therefore, they are mobile and changing.
3. They appear in both stages of breathing, especially in expiration.
4. They give rise to a characteristic palpable vibration called bronchial thrill, which we already studied.
5. They are not affected by pressure, but are modified or disappear by breathing, coughing and expectoration.

6. They must be differentiated from large dry friction, whose characteristics have already been studied.

Semidiagnosis

1. They are frequently observed in the initial period of acute bronchitis accompanying wheezing rales and are later replaced by wet rales.

2. They also occur in the dry forms of chronic bronchitis.

3. Finally, in bronchial compressions or obstructions of different nature and location.

B. Wheezing rales (see Fig. 34.10).

Semiogenesis or pathophysiology

They are caused by the narrowing of the bronchial lumen in the fine bronchi, due to viscous or adherent secretions. Also, due to spasm of the Reisseisen muscles and edema in the fine bronchi as happens in asthma at the beginning of the crisis.

Sometimes both factors are added, as observed in severe asthma attacks.

semiography

1. Continuous high-pitched noise that reminds:

a) The hiss of air when it violently passes through the crack of a door.

b) The singing of some birds.

2. They are heard over the entire surface of the chest and their intensity sometimes allows them to be heard from a distance, by the patient himself and those around him.

3. They are mobile and changing, although less than

hoarse

4. They appear in both stages of breathing, especially in expiration.

5. Sometimes bronchial thrills can be palpated.

6. They are not modified by pressure, but they are modified by coughing, breathing and sometimes they disappear with expectoration.

7. Their diagnosis is generally imposed, since it is very difficult to confuse them with other noises.

Semidiagnosis

These rales commonly appear in:

1. Asthma. Due to spasm of the Reisseisen muscles accompanied by mucosal edema and viscous and adherent secretions.
2. Acute bronchitis. In the period of crudeness when they reach the fine bronchi.

3. Dry forms of chronic bronchitis.

4. Compressions or obstructions of fine bronchi.

Moist, mucous, or bubbly rales A. Crackling rales.

Semiogenesis or pathophysiology

Production mechanism. The crackling rattle is the alveolar rattle par excellence. Three causes can determine it:

1. The detachment of the alveolar walls from the fibrinoleukocyte molds that fill them, as occurs in pneumonia.

2. The mobilization with inspiration of fluid alveolar transudates as occurs in heart failure and with greater intensity in acute pulmonary edema, where due to its ascending and rapid installation from the base upwards it has earned it the picturesque name of rising tide rales, or mount. Sometimes they are associated with fine subcrackle rales.

3. The displacement of the collapsed alveolar walls as occurs in atelectasis, in this case called unfolding rales.

The old view that crackles came from fluid in the airways has been replaced by another explanation, according to the results of research carried out by Forgacs and other scholars.

Currently, it is considered that these rales are caused by the sudden and successive opening of the small airways that had closed prematurely during expiration, which allows the pressures above and below the obstruction to equalize and is translated sonorously as mini-explosions.

These phenomena, premature closure of the small airways during expiration and sudden opening during inspiration, are associated with inflammatory processes with fibrinoleukocyte exudates, as in pneumonia, and with congestion and pulmonary edema, as in heart failure.

These morbid processes can cause disorders in the lung's dilation capacity and elastic retraction.

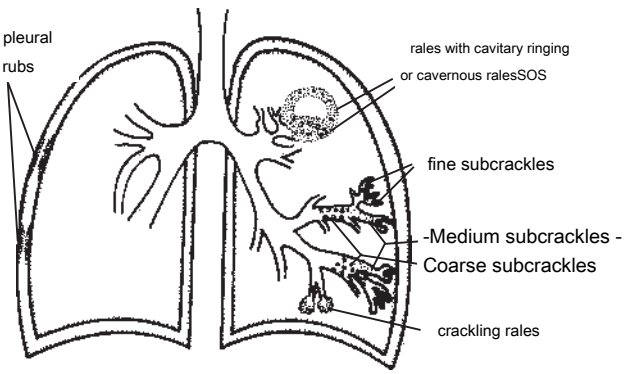
semiography

First variety of wet rales; They have their own characteristics (fig. 34.11):

1. They are short, small and fine noises, equal to each other in intensity and duration, regularly spaced. They are compared to the noise of salt crackling with gentle heat in an iron vessel, to the noise produced by a dry bladder when inflated, to the noise obtained when squeezing healthy lung tissue filled with air with the fingers. They are imitated by rubbing hair near the ear. They seem to project themselves into the ear like bursts of small rockets.

Fig. 34.11 Crackling, subcracking and crackling rales

cavernous.



They give the sensation of great dryness, but sometimes they appear moist and less regular as occurs in acute pulmonary edema.

2. They are perceived at the site where the lesion appears and although they disappear for some time, they always reappear in the same place, which gives them diagnostic value.

3. The moment of breathing in which they appear is important. They are heard only on inhalation, sometimes only in the second half or last third of it.

4. They do not give palpatory sensations.

5. They do not suffer modifications due to pressure, breathing, coughing and expectoration; they disappear for brief moments; They reappear at the same point.

6. The diagnosis must be established against:

a) Fine subcrackle rales. These are differentiated by being thicker, irregular, more mobile bubbles, modifiable by breathing, coughing and expectoration and audible in both stages of breathing.

b) The friction-rattles. They are more difficult to differentiate, but they are heard at the end of inspiration and the beginning of expiration.

Semidiagnosis

1. Pneumonia. The typical crackling rattle appears with great dryness. It occurs early at the beginning of pneumonic condensation. When condensation has established itself, that is, in the state period, it gives way to the tubal murmur that is surrounded by a crown of crackling rales. At the end of the process they appear again, being thicker, moist, irregular, located throughout inspiration, and are called return crackles.

The crackling rattles of pneumonia begin to be heard in the second part of inspiration and return sounds throughout the inspiration.

2. Bronchopneumonia.

3. Edema or pulmonary stasis (acute and chronic heart failure).

4. Corticopleuritis.

5. Brouardel's unfolding rattles. They appear at the base of the lung when the subject stands up and are due to the deployment of alveoli that were atelectasis due to the compression of recumbency; It is important to take them into account, especially in patients who remain in the supine position for a long time, since they in themselves have no pathological significance.

They disappear after several inspirations.

B. Subcrepitating rales (see Fig. 34.11).

Semiogenesis or pathophysiology

They are caused by the bursting of air bubbles in the secretions of the bronchial lumen or by the collision of these secretions and the air currents of the respiratory tree. They appear whenever there are fluid secretions in the bronchi, which may come from the bronchi themselves, from other pathological processes of the thorax or abdomen that have made their way to the bronchial lumen.

semiography

1. They are called mucous rales; They give the sensation of great humidity. They are heard irregularly spaced, thicker and more uneven than crackles. They are divided according to the thickness of the bubbles into fine, medium and thick:

   a) Thick. They resemble the noise produced by blowing an absorber into a container full of water.

   b) Fine. They are sometimes confused with crackles.

   c) Medium. They are intermediate between the previous two.

   The importance of this classification lies in the fact that the thickness of the bubbles is closely related to the caliber of the bronchus in which they occur.
2. The place where they appear is very variable; Therefore, They are very mobile and changing.
3. They appear in both periods of breathing, especially in expiration.
4. Only in exceptional conditions do they give palpatory sensations.
5. They do not undergo variations due to pressure, but coughing and expectoration make them appear or disappear.
6. The diagnosis must be established between fine subcrepitants and crackles. Subcrackles are more humid, irregular and changing, they are not fixed. They are heard during both breathing periods and are modified by coughing and expectoration.

Semidiagnosis

The conditions in which they usually appear are:

1. Hemoptysis.
2. Evacuation of pleuropulmonary, mediastinal or abdominal abscesses by vomiting, or drainage of suppuration from the bronchi, lungs or pleura.
3. Acute bronchitis during cooking period. If in these cases we hear thin subrepitants, we must presume the imminence of alveolitis, that is, bronchopneumonia.
4. Pneumonia in the process of resolution.
5. Bronchopneumonia.
6. Pulmonary tuberculosis. This disease gives the typical subcrepitant rales more frequently, since in tuberculosis there may be bronchial inflammation, softening of caseous nodules, hemoptysis, suppuration, etc.; that is, a whole series of pathogenic mechanisms capable of generating these death rattles.

C. Cavernous rales (see Fig. 34.11).

Semiogenesis or pathophysiology

It is the same as the fine subcrepitant, but the thick bubbles inside a cavity give it the cavernous timbre that accompanies the cavity murmur. The cavity can also be a thick bronchus with secretions of varied origin, as occurs in pseudocavitary syndrome, especially in the interscapulovertebral space.

semiography

1. They are subcrackling rattles of thick bubbles, like a moist click that accompany the cavernous murmur; They give rise to a complex noise called gurgling.

   These bubbles are heard isolated, sometimes they disappear due to coughing and reappear after the cavity is filled.
2. They are located at a fixed point, where the determining cavity lesion is located.
3. They appear in both times of breathing; They disappear after abundant expectoration.
4. They do not give palpable sensations.
5. They do not change due to pressure, but they do change due to coughing and expectoration that make them disappear.
6. They are unmistakable with any other rattle, due to their characteristics already mentioned, especially because of the hollow timbre and because they are accompanied by a cavernous murmur.

Semidiagnosis

The conditions in which they usually appear are:

1. Tuberculous caverns.
2. Bronchiectasis.
3. Lung abscesses.
4. Pulmonary gangrene.
5. Evacuated cystic pleurisies.
6. Pseudocavitary syndrome.

AUSCULTATION OF COUGH

The value of cough in auscultation is mainly given by: acoustic and physical characteristics, and its contribution to the diagnosis of other auscultatory signs.

Acoustic and physical characteristics

They vary with the physical conditions created by the pleuropulmonary injury. So we have:

a) Cavernous cough, which appears in tuberculous cavitaries and gives the sensation of tearing.

b) Dry, dull cough that appears in pleuritic patients.

c) Harsh or barking cough (barking cough), which appears in tracheobronchial stenosis.

d) Amphoric cough, with a metallic ring, heard in pneumothorax.

Contribution to the diagnosis of other auscultatory signs

They occur because cough can:

a) Intensify respiratory sounds, facilitating their study.

b) Evidence of blowing respirations and light murmurs such as pleuritic murmurs.

c) Make subcrackle and cavernous rales disappear.

us.

VO AUSCULTATION

Concept and semiog nesis

The vocal sounds produced by the vibration of the strings are modified in their intensity, tone and timbre by the air spaces located above the larynx (mouth and nose) and by the trachea, bronchi, lungs and thorax.

They are governed by the same principle of vocal vibration, therefore, letters and syllables are normally not heard due to the overtones added to the fundamental tone that characterizes most sounds, and which are greater than 170 vibrations per second.

The vibrations are more intense in men than in women, and undergo modifications due to age, region, wall thickness, etc.

They have their origin in vibrations originating in the vocal cords and propagated by the air to the chest wall.

Semiography

The auscultation of the natural voice and the whispered voice (voiceless pectoriloquy) must be carried out using the word 33, which is rich in R, a letter that has great vibration.

Normal resonance of the natural or normal voice

The voice is heard as an indistinct murmur in which no syllables or words can be distinguished.

Semidiagnosis. There may be decreased resonance and increased resonance. Pathologically the processes laryngeal and pleuropulmonary can determine a decrease or increase in the intensity of the voice, occasionally making it possible to distinguish letters and syllables.

1. Decrease or diminished resonance. It appears in processes that attenuate or make vocal vibrations disappear, such as: bronchial obliterations (obstructions), emphysema, pleural effusion, pneumo-

chest.

2. Increased resonance. It appears in all the processes that increase vocal vibrations, such as:

lung condensations, cavities.

In both cases the bronchial tree must be patent.

3. Exaggerated resonance of the voice. It is the pathologically increased intensity that acquires greater value when it is localized. It has the same significance as bronchovesicular or blowing respiration. It therefore appears in low-volume pulmonary condensations that do not cause either tubal murmur or bronchophony.

4. Bronchophonia. It is equivalent to the tubal murmur. Very high degree of exaggerated resonance of the voice without distinguishing syllables.

They appear in: pulmonary condensations that reach 3 mm bronchi, such as: pneumonia, bronchopneumonia, pulmonary congestion, tuberculosis, tumors, etc.

5. Pectoriloquy or cavernous voice. It accompanies the cavernous breath; It is more serious and intense than bronchophonia and acquires a special timbre where letters and syllables are heard, giving the sensation of speaking from the bottom of the chest, hence its name.

In the lung cavities it can be intense and causes an unpleasant sensation in the ear. It appears in superficial lung cavities of medium volume: tuberculous cavity, bronchiectasis, abscess, pseudocavity. Lung condensations that reach deep into bronchi 6 mm in diameter can also cause it.

6. Amphorophony or amphora voice. The physical conditions that produce the amphoric murmur make amphorophonia appear, which is nothing more than a metallic resonance of the voice whose metallic tone is similar to that produced when speaking into an empty amphora.

7. Egophony. Voice of a goat or a polichinella. Its stetoacoustic characteristics include modifications of the auscultated voice, which becomes trembling, with a sour timbre and a very high pitch. The trembling voice is compared to the bleating of the goat and hence its name, egophony:

goat voice

It remembers the voice of the ventriloquist, hence its name puppet voice or puppeteer.

It has been shown to appear in:

Pleural effusions of any nature but of medium intensity where they accompany the pleuritic murmur at its upper limit. It disappears in the big de-

ITames.

Hydrothorax.

Splenopneumonia.

Corticopleuritis: where it is heard more intensely due to the existence of a laminated effusion.

Pleuropneumonia: where it mixes with bronchophonia, giving bronchoegophonia.

Voiceless pectoriloquy

The chest is listened to when the patient speaks in a whispered or hoarse voice, as if secretly saying 33.

Semiogenesis and semiography. In a normal state, a more intense indistinct murmur is heard near the thick bronchi and at the right vertex; There are cases in which nothing is heard.

In voiceless pectoriloquy, when the patient speaks, the letters, syllables and words are heard, giving the sensation of speaking secretly inside the thorax.

It is due to Bacelli of Rome, hence the name Bacelli sign. This author described it as typical of pleurisy with serofibrinous effusion, not of purulent and hemorrhagic pleurisy in which leukocytes and red blood cells would interfere with the transmission of sound-producing waves.

Semidiagnosis. It has currently been shown to appear in:

1. Pleural effusions of any nature.
2. Pulmonary caverns.
3. Lung condensations: bronchopneumonia, pneumonia, etc.
4. Splenopneumonia.
5. Pneumothorax.
6. Tracheobronchial lymphadenopathy: in this case they are heard in the spinal column and are called whispered voice or D'Espine sign.

PITRES COIN SIGN

This sign has a great diagnostic value in determining the amount of liquid in a spill, since through it the height of the spill is obtained. To investigate this, the chest is listened to at the same time as an assistant strikes with two coins, one against the other placed at the same height, but on the opposite side of the chest.

The sign is positive when the metallic sound clearly reaches the ear of the observer, in such a way that it seems as if these coins were hitting close to the ear of the observer, who perceives a clear and silvery sound. When the sign is negative, the listener perceives a dull, dull, brief noise devoid of metallic timbre, similar to the sound of wood percussion, characteristic of normal lung parenchyma.

Semiodiagnosis

Appears in:

1. Pleural effusions.
2. On normal occasions it can be heard below the tenth rib in the posterior plane, hence it only reflects pathology above this rib.

35

RESPIRATORY SYSTEM. EX MENES

COMPLEMENTARY

After carrying out a careful clinical examination of the respiratory system, we are in possession of a good number of symptoms and signs that allow us to form different respiratory syndromes. In many cases, only with these elements can we accurately establish a diagnosis and precise therapy, such as asthma, heart failure, pneumonia, pneumothorax, etc. However, on other occasions, either because the symptomatic elements are not yet very conclusive - as happens in the beginning periods of some respiratory diseases - or because the deep situation of them in the lung does not show symptoms, or because we cannot accurately define the magnitude of the process and its etiology, we are obliged, whenever possible, to use the help of a group of auxiliary examinations without forgetting some important principles:

1. Do not abuse them unnecessarily.
2. Know how to appropriately weigh the usefulness that the examination we request will provide to the patient and the degree of discomfort or risks that it may cause.
3. Proceed according to a philosophical principle "from the simple to the complex";

This means that, sometimes, with some laboratory tests, including sputum and a simple radiological examination, we can obtain enough information to corroborate our diagnostic impression and institute appropriate therapy without the need to use bronchography, a pleural biopsy or a mediastinoscopy, for example.

The most commonly used examinations in the respiratory system are:

- Clinical laboratory investigations.
- Study of expectoration.
- Examination of gastric contents.
- Bronchial lavage.
- Exams that must be performed with the material obtained in expectoration, gastric contents and bronchial lavage.

Electrocardiogram.

- Functional examination of the respiratory system.
- Pleural and lung biopsies.

- Endoscopic methods:

Bronchoscopy.
Thoracoscopy.
Mediastinoscopy.

- Radiological and imaging examinations:

Radioscopy or fluoroscopy.
Simple radiological examination.
Simple or conventional tomography.
Bronchography.
Computed axial tomography.
Nuclear magnetic resonance.
Pulmonary angiography.
Lung scintigraphy.
Ultrasonogram.
Echocardiogram.
Image-guided percutaneous biopsy.

We will limit ourselves here to pointing out its indications and contraindications as briefly as possible.

CLINICAL LABORATORY INVESTIGATIONS

In some respiratory tract conditions, a diagnosis can only be reached through laboratory investigations, which gives an idea of the importance they have for the practical doctor. Hemogram, blood glucose, erythrocyte sedimentation rate and others are routinely indicated.

STUDY OF EXPECTORATION

Sputum

Of all the investigations that could be carried out on a patient classified as having pulmonary disease, the most important is that of sputum.

The collection of the material is of the greatest importance; The patient will be recommended to obtain a sample of the contents of the bronchial tree through coughing, or from the larynx through tearing. Sometimes, when pulmonary suppuration is suspected, the entire 24-hour sputum is collected to evaluate its macroscopic appearance.

When it is necessary to investigate the bacteriological content, a fresh sample of sputum must be collected, because if it is delayed in its examination, saprophytic bacteria grow and make the diagnosis difficult. The sample should always be collected in a clean, preferably sterile, bottle.

EXAMINATION OF GESTRIC CONTENT

As appreciable quantities of expectoration tend to be swallowed imperceptibly, it is often advisable to examine the gastric contents obtained by tubaje. This sample must be seeded quickly to avoid bacterial destruction by digestive juices.

The material is obtained early in the morning and this investigation is generally used to demonstrate the presence of the tubercle bacillus.

BRON UIAL WASH

It consists of the introduction of approximately 5 mL of saline solution into the trachea with the purpose of provoking a violent cough and collecting the expectorated material.

It is recommended during bronchoscopic examinations to use this system to obtain a sample specifically from areas suspected of injury, and collect the fluid through a suction cannula fixed in a collection tube.

It is also advisable to collect sputum that appears within 24 hours after a bronchoscopic investigation, for examination.

EX MENES UE SHOULD BE CARRIED OUT WITH THE MATERIAL OBTAINED IN THE EXPECTORATION, THE GESTRIC CONTENT AND THE BRON UIAL WASHING

We must consider that in the case of a suspected case of pulmonary tuberculosis, the following investigations should be carried out:

EXTENSION AND CONCENTRATION OF SPUTUM FOR COLORATION AND DIRECT EXAMINATION AND OMOGENEI ADO

The technique is staining for acid-fast bacillus (Ziel-Nelssen).

SPUTUM CULTURES

They can be performed both to check for Koch bacillus and to assess its resistance to anti-tuberculosis drugs.

The certainty diagnosis of tuberculosis is of such importance that culture should always be used, even in those cases in which abundant bacilli have been obtained in direct examination, but in which the diagnosis is established for the first time.

INOCULATION TO CURIEL

Although current culture methods can be compared in effectiveness with curiel inoculation, this latest research maintains the advantage that truly virulent organisms are the only ones capable of multiplying; This eliminates the risk of false positive reactions.

CYTOLOGICAL STUDY OF SPUTUM

Bronchial carcinomas very commonly shed malignant cells with sufficient characteristics to be able to establish their diagnosis. The absence of these cells, however, does not allow us to exclude the possibility that it is a tumor of this nature.

EXAMINATION OF SPUTUM BY THE GRAM METHOD

Slice extension and Gram staining provide additional help to culture techniques, especially in the study of pulmonary suppurations. There are germs such as Klebsiellay Haemophilus that are difficult to culture, but the diagnosis is quickly established using the Gram method.

ANTIBIOGRAM ANTIBIOTICOGRAM OF SPUTUM

It is frequently convenient to establish, in the face of a certain suppurative condition of the lung, what type of antibiotic would be the most appropriate or effective. For this purpose, a suspension of sputum or bronchial content is sown in one or several culture plates to which small discs or tablets containing the antibiotic that we are going to test are added. The zone of inhibition of bacterial growth around the disc allows us to establish a rough concept of the degree of sensitivity of the bacterial flora to the antibiotic.

BS UEDA OF NGOS IN THE SPUT0

The isolation and identification of fungi in sputum constitutes an extraordinarily complicated and difficult procedure. Some fungi can be more easily identified by their macroscopic appearance in sputum; To do this, it is placed on a glass sheet with a coverslip and exposed to light, which allows granules or masses of filaments to be seen, characteristic of some of these infections, such as, for example, actinomycosis.

RESEARCH FOR THE DIAGNOSIS OF VIRAL CONDITIONS

It requires the collaboration of a highly specialized laboratory and the help of an expert biologist. Some viruses are isolated from sputum, as in psittacosis and ornithosis (contact with birds, flowers, food for these birds, etc., very important in the diagnosis), others by inoculation of the nasopharyngeal lavage to mice or in the amniotic sac of the chicken embryo, as occurs with influenza A, B and C. But in other viral conditions, the diagnosis is made by immunological methods in the blood (complement deviation, antibodies, etc.), or by isolating cold agglutinins, as occurs in atypical viral pneumonias.

EOSINOPHIL COUNT IN SPUTUM

This indication is common in cases of bronchial asthma where a large number of these cells appear in the sputum spread. This does not imply that there is parallel eosinophilia in the blood.

When faced with pulmonary images suspicious of Loeffler syndrome or tropical pulmonary eosinophilia, the absence of these cells in the sputum completely rules out the diagnosis.

ELECTROCARDIOGRAM

It is a test of great importance in respiratory tract conditions, since it is important to assess possible right-sided insufficiency in conditions such as pulmonary emphysema, pulmonary fibrosis, pneumoconiosis, etc., which leads to a condition called chronic cor pulmonale. We also find it essential to use it in the preoperative phase of surgical conditions of the pulse.

mon.

FUNCTIONAL EXAMINATION OF THE RESPIRATORY SYSTEM

The fundamental function of the respiratory system is to ensure the gas exchange of oxygen and carbon dioxide between the alveoli and the wall of the pulmonary capillaries. Oxygen passes from the alveoli to the capillary blood, due to the greater tension in them;
The opposite occurs with carbon dioxide, since its partial tension is greater in the pulmonary capillary. This function that finally ensures gas exchange is carried out in the following phases:

1. Ventilation.

2. Air distribution.

3. Pulmonary capillary blood flow (explainable by the functional autonomy of the lung).

4. Alveolocapillary diffusion or hematosis.

We will not go into describing the tests that are carried out to study each of these aspects, which can be seen in figure 35. 1.

BRIEF PRACTICAL DESCRIPTION

Respiratory function tests are very useful in the initial and evolutionary diagnostic assessment, in such a way that they offer us a measure of the response to treatment and its clinical evolution. They generally have greater sensitivity than other studies to detect alterations of the airways and lung parenchyma, even without the presence of symptoms.

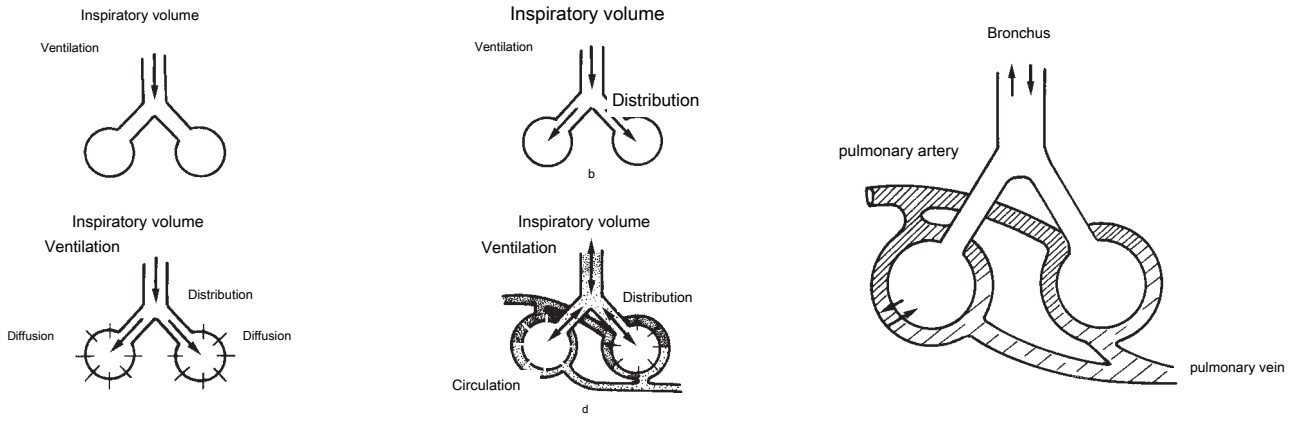


Fig. 35.1 Partial respiratory functions: a, ventilation; b, ventilatory distribution; c, diffusion; d, alveolocapillary gas exchange (According to Comroe.) Ventilation depends on the amount of parenchyma, thoracic mobility and bronchial permeability. Diffusion depends on the permeability of the alveolocapillary membranes. Circulation depends on cardiac output and the caliber of the capillaries that govern pulmonary pressure.

The basic diagnostic test for lung function is spirometry. For this purpose, instruments called spirometers are used, generally water or bellows or with electronic devices that allow air flow to be determined.

The patient, after a series of tidal volume ventilations, must inhale deeply until full lung capacity is achieved. Then, you must quickly exhale as much as possible until you reach the residual air level. The results of the forced expiratory maneuver can be represented as a time-volume relationship, in which the total number of liters exhaled is represented on a vertical axis and the elapsed time on the horizontal axis.

Among other indices, forced vital capacity and maximum expiratory volume in one second are determined.
The results are compared with the established theoretical values, based on the age, height and sex of the subject.

Another more recent way of representing the expiratory maneuver is the flow-volume curve.

There are other specialized tests to measure other aspects of lung function, such as determining residual volume and diffusion, but they are beyond the scope and objectives of this work.

Yes, we are going to briefly point out the importance of hemogasometry, particularly the measurement of arterial pH values, arterial oxygen pressure (paO,) and carbon dioxide pressure (paCO,), for the diagnosis of disorders that compromise lung function.
For example, hypoxemia may be related to hypoventilation, pulmonary diffusion abnormalities, cor-right-left tocircuit, alterations in the ventilation-perfusion relationship and alveolar hypoxia.

It is worth clarifying that hemogasometry is also useful in the diagnosis of alterations in acid-base balance. In it, not only arterial values are determined, but also capillary and venous values.
In addition to paO, paCO, and pH, other parameters are studied such as standard bicarbonate (BS), base excess (BE) and hemoglobin saturation (HbO,).

REFERENCE VALUES OF SOME PAR METERS ei| C il

p	7.35-7.45 35-	
pacO, (mm 9)	45	
pa O, (mm g)	95-100	75-100

Intensive care. Cuban Assistance Standards, 1977.

ashington manual, 9th. edition, 1996.

PLEURALY AND PULMONARY BIOPSIES

They can be performed through small thoracotomies, thoracoscopy or using a needle or trocar designed for this purpose. They are indicated in lung or pleural lesions of a certain size and whose diagnosis has not been established.

M ALL ENDOSCOPIC

BRONCHOSCOPY

Bronchoscopy is an indispensable auxiliary means of exploration in respiratory diseases.

It allows direct observation from the vocal cords to the segmental bronchi, allowing us to obtain not only the vision of the pathological alterations existing along this path, but also the obtaining of biopsies or secretions for bacteriological or cytological examination and on occasions it has been used for the direct treatment of bronchial lesions through touches, instillations, fulgurations, etc.

Indications

Pulmonary tuberculosis is a frequent cause of bronchoscopic examination, due to the location of the lesions at the level of the entrance orifices of the segmental bronchi, making it possible to establish the diagnosis and perform local treatments.

Bronchial carcinoma is another condition that requires bronchoscopic examination, since 50% of these neoplasms can be diagnosed through said exploration, which also allows us to obtain the corresponding sample or biopsy for microscopic examination. When the existence of the lesion is not proven, the secretion can be taken for examination, which in most cases allows us to specify the possibility of tuberculosis or neoplasia.

This procedure is also used to check where purulent secretions or hemorrhages come from in patients with few radiological symptoms.

It can be performed for therapeutic purposes, such as aspiration and/or bronchial lavages.

Contraindications

They are very scarce. For large hemoptysis, it is advisable to wait a few days to perform the exam. A few years ago, some patients with rheumatic disorders of the cervical vertebrae or with prominent teeth, or individuals with short and thick necks in whom the exploratory act used to be extraordinarily difficult, were contraindicated. In aortic aneurysms it was extremely dangerous due to the risk of rupture and death of the patient.

In old or very weakened patients, the investigation is deferred, since the age or conditions of the patient do not allow it. Currently, with fiberoptic bronchoscopes that are not only very thin but totally flexible, this examination has gained great relevance and has made it easier to reach small bronchi without complications or risks, since they have movements at their distal end that allow them to be guided without difficulty. Through them we can perform biopsies, aspirations, photographs, electrocoagulation, etc.

THORACOSCOPY

Through it, visual exploration of the pleural cavity is carried out through the introduction of an optical system. With the advancement of technology, the instruments have been perfected and are currently made with a video-endoscopic system. It has few contraindications, except in the case where a pleural biopsy is also performed. Among its main indications are the study and treatment (pleurodesis) of pleural effusions;

endoscopic evaluation and treatment of pneumothorax;

lung biopsy in diffuse interstitial pneumonia;

study of peripheral lung lesions; chest wall lesions and mediastinal tumors.

MEDIASTINOSCOPY

It is actually a surgical technique in which, through an incision in the suprasternal cavity, an optical instrument is introduced to explore the upper portion of the mediastinum and nearby regions. It requires general anesthesia and is often complemented by an anterior mediastinotomy. It has to be carried out by experienced personnel and is not free of risks and complications. The main contraindication is, among others, "superior vena cava syndrome" and its most important indication is the detection of possible lymphadenopathy in patients with bronchopulmonary cancer, to decide whether it is resectable or not.

RADIOLOGICAL EX MENES

This subject is the subject of study in another subject, so we will only point out very general aspects of the same.

The respiratory system is possibly one of those that benefits the most from radiological assistance; For this, multiple techniques are available. We will mention among them:

- Radioscopy or fluoroscopy.
- Simple radiological examinations.
  In frontal, lateral, oblique, vertex and preparation techniques.
- Simple or conventional tomography.
- Bronchography.
- Angioneumography.
-Ultrasonography
- Echocardiogram
- Lung scan Image-guided biopsy

RADIOSCOPY OR FLUOROSCOPY

It is practically discarded at the present time, as it is a technique that has many drawbacks;

The main one is the X-ray dose absorbed by both the patient and the observing doctor.
Nowadays, when the aid of television is available, through an image intensifier that uses a minimum of milliampere, this problem is eliminated at least in the aspect concerning the medical personnel, who logically are the ones that receive the greatest number of radiations, due to the need to examine numerous patients.

It is the only radiological method that allows us to study the functioning of the thoracic organs and to specify, through the movement and change of positions of the patient, the location of the lesions, determining, as far as possible, the techniques and indications of the remaining radiological studies.

SIMPLE RADIOLOGICAL EX MENES

They allow the use of various techniques. As a basic element, it is always advisable to use the front and side plates together to be able to exactly locate the injuries. The right and left anterior oblique positions (OAD and LAA, respectively) are of greatest interest in cardiovascular conditions. Likewise, so-called vertex plates are used, with the patient in a position of exaggerated kyphosis and lordosis, in order to displace the bone elements that cover the upper lung extremity and be able to adequately visualize the alterations of the vertex.

Finally, it is sometimes possible to determine the characteristics of certain lesions with greater penetration than that corresponding to the simple plate.

SIMPLE OR CONVENTIONAL TOMOGRAPHY

It allows you to analyze a frontal or lateral plane section of the thorax, located at a certain depth, without images of the other planes appearing. The selective image is generally obtained by simultaneously moving the X-ray tube and the plate in reverse direction, with the chosen section constituting the axis of movement. It is a widely used method against imprecise images in chest conditions,

Despite the advent of computed axial tomography (CAT), conventional tomography retains its usefulness and indications, especially when it is not available. It is very sensitive for the study of metastatic pulmonary nodules, as well as useful for the characterization of solitary pulmonary nodules. It also reveals diagnostic precision in the bronchovascular structures of the pulmonary hilum.

BRONCHOGRAPHY

It constitutes the radiological exploration of the bronchial tree; In it, contrast substances are used that provide They give an opaque image of the bronchial tract. It requires taking views in various positions to adequately identify the affected bronchial branch, its alterations, location and degree of pulmonary compromise that causes the condition.

COMPUTER AXIAL TOMOGRAPHY ADA TAC

The basis of this modern diagnostic technique is the emission of a beam of ionizing radiation that passes through the patient in a circular manner. The radiation is collected by a series of multiple detectors that a computer transforms into digital images. Computed axial tomography (CAT) has meant a great leap in quality in the diagnostic evaluation of lung diseases. With the use of contrast and current technology, its diagnostic possibilities cover a spectrum of chest conditions, ranging from the chest wall to the pulmonary vasculature.

NUCLEAR MAGNETIC RESONANCE NMR

This novel imaging method is based on the behavior of protons between magnetic fields and radiofrequency waves. It has an advantage over other imaging techniques due to: the achievement of a high contrast between the different tissues, the absence of ionizing radiation, the possibilities of directly obtaining multiple planes, without the need for reconstruction, as well as intravascular observation without contrast.

Despite this, its indications in thoracic pathology are still limited. Its great value is recognized in the study of the posterior mediastinum, the neural canal, the great vessels, the evaluation of Pancoast tumor and parietopleurodiaphragmatic diseases.

Disadvantages include its high economic cost, which makes it available in very few hospital centers, prolonged examination time and poor visualization.
calcium tion.

PULMONARY ANGIOGRAPHY

It is a method that allows the opacification of the arterial and venous circulatory system, at the level of the respiratory system, by introducing a radiopaque substance.

Serial radiographs must be taken, that is, at short time intervals, to obtain the progression of the material through the vascular bed. Its main indications are: the evaluation of patients with suspected pulmonary thromboembolism and to determine the existence of arteriovenous fistulas. Bronchial arteriography has its main indication and diagnostic and therapeutic usefulness in severe hemoptysis.

Currently, in addition to the conventional technique, in which the image obtained is analog, the digital subtraction method is available.

PULMONARY SCAMMAGRAPHY RADIOACTIVE ISOTOPES

Although it has its main utility in the diagnostic strategy of thromboembolic disease, the use of radioactive isotopes is also indicated in the study of pulmonary capillary permeability, and in adult respiratory distress syndrome, as well as in the diagnosis of pulmonary pyogenic infectious processes and the evaluation of diffuse infiltrative diseases in the acute phase (sarcoidosis, AIDS, idiopathic pulmonary fibrosis).

Among the radioactive isotopes, used by inhalation, intravenous or jointly, are Technetium 99m (99,), Xenon (133, and 127,,) and Gallium 67 citrate (67), among others.

ULTRASONOGRAPHY

It is another method currently under development, which has the advantage of not using radiation and allows all regions of the thorax to be easily observed by simply moving the small device that is in the radiologist's hand and that projects the image on a screen, allowing photographic images of the lesions to be obtained. It also allows you to observe the functioning of the bronze tree.

copulmonary It is useful in small effusions, cysts and tumors.

ECHOCARDIOGRAM

Cardiac diagnostic ultrasound using Mode M and Mode B allows us to recognize the impact of respiratory conditions in the right heart chambers (chronic cor pulmonale). The use of cardiac Doppler makes it possible to detect pulmonary hypertension.

IMAGE-GUIDED PERCUT NEA BIOPSY

With the help and guidance of an imaging technique, be it conventional radiology, ultrasound or CT, a needle is introduced and the lesion is punctured.

According to the diagnostic objectives and the dimensions of the needle, a sample is taken for cytology (aspiration biopsy, or aspiration cytology, with fine needle, FNAB) or for histology (with thick needle or Trucut-type needle-trocar). Its main indication, among others, is the diagnosis of the nodule that cannot be reached through the endotracheal fiberendoscope.

36

The grouping into syndromes of the different symptoms and signs previously studied facilitates, and sometimes allows establishing, the nosological diagnosis of a condition of this system. It also shows us the value of the semiology studied in the previous chapters and constitutes the true essence of the clinic, which will never disappear, nor can it be replaced by technology.

We will consider:

- Bronchial syndromes.
- Pulmonary syndromes.
- Pleural syndromes.

Mediastinal or mediastinal syndrome.

- Respiratory failure syndrome.
- Adult respiratory distress syndrome or adult type.

BRON UIIAL SYNDROMES

When inflammation or obstruction of a bronchus occurs due to spasm, aspiration of a foreign body, or any other cause, of course, a decrease in the caliber of the bronchial lumen occurs, which results in difficulty in the entry or exit of air, in which case it can cause the formation of emphysematous vesicles, if the bronchus is not completely obstructed, or atelectasis, if the obstruction is total.

Among the bronchial syndromes we will treat:

- Acute bronchial syndrome. Acute bronchitis.
- Chronic bronchitic syndrome. Chronic bronchitis.
- Bronchial obstructive syndrome.
- Bronchial asthma syndrome.
- Bronchiectasis syndrome.

ACUTE UITIC BRON SYNDROME. BRON ACUTE UITIS

Concept

It originates from acute inflammation of the bronchial mucosa, due to various causes (infectious, irritative, allergic) and is almost always accompanied by inflammation of the tracheal mucosa (tracheobronchitis).

Syndromography or positive diagnosis

Clinical picture

a) Initial or raw period.

Pharyngeal tickling, dry, irritating cough, which may present in the form of cough quints. Absent or scant expectoration. Retrosternal gravity discomfort that when exhaling deeply gives a sensation of roughness or injury. Few general symptoms or discomfort, low-grade fever and anorexia.

b) State period.

After two or three days, the cough is less bothersome, it becomes wet, expectoration appears, which causes a feeling of well-being, and retrosternal discomfort decreases. The initial sputum is mucous and transparent. They then become mucopurulent, thick, greenish-yellow in color, which are easily expelled.

c) Physical examination.

Inspection: normal.

Palpation: occasionally, bronchial thrill, especially due to hoarse rales and normal vocal vibrations.

Percussion: normal lung sound.

Auscultation: normal or harsh vesicular murmur.

Dry hoarse and wheezing rales, scarce in the initial phase and then abundant, in both hemithoraxes. Already in the state period, subcrackling rales, especially thick and medium-sized, may appear, especially at the bases, in relation to the mobilization of secretions. Auscultation of normal voice.

Complementary exams

Chest x-ray. No alteration is observed or only a reinforcement of the lung structure in the form of a network or narrow meshes due to congestion of the vascular network, which goes to the vertices and bases to form the upper and lower hilar horns.

Syndromogenesis and etiology

- Primary acute bronchitis, caused by various bacterial and viral agents.
- Acute bronchitis secondary to a local condition: cold, rhinopharyngitis, sinusitis, tonsillitis (focal tracheobronchitis) or due to a general condition, such as measles and typhoid fever.

Tracheobronchitis due to inhalation of irritating dusts.

CHRONIC BRON UITIC SYNDROME. BRON UITIS CHRONICLE

Concept

Clinical syndrome caused by chronic inflammation of the bronchial mucosa, the starting point of various infectious and irritating factors.

Syndromography or positive diagnosis

Clinical picture

Symptoms:

Cough, which predominates in the morning and in advanced cases becomes constant and painful, causing insomnia and fatigue; generally productive and with a gravey, bubbly tone.

Mucopurulent expectoration, with globular, greenish-yellow or greenish-gray, odorless sputum.

In some forms, it can be downright purulent and in rare cases, fetid. In other cases, the expectoration may be mucous, very stringy and abundant. On the other hand, in the so-called dry forms, expectoration may be very scarce.

Dyspnea, a fundamental symptom in chronic bronchial patients, which ranges from a feeling of discomfort or certain respiratory distress in periods of exacerbation, to dyspnea on exertion, with intercurrent paroxysmal attacks of an asthmatic type or to a morning obstructive form, which subsides after the expulsion of thick and compact sputum. If this condition persists, it can cause pulmonary and cardiac complications (chronic cor pulmonale) and even recumbent dyspnea.

Physical examination:

General inspection. There may be hypertrophic pneumoarthropathy in very old cases. Facies: in very advanced cases, with chronic airflow limitation, the "blue bloater" facies can be seen.

Respiratory exam:

Inspection: chest appears normal, OR barrel-shaped if associated emphysema is present.

Palpation: normal or decreased vocal vibrations. Bronchial fremitus due to snoring.

Percussion: normal lung sound, or increased if there is associated emphysema.

Auscultation: harsh vesicular murmur, which may be diminished, depending on the abundance of rales. Hoarse and wheezing rales scattered in both hemithorax. Subcrackle rattles of large and medium-sized bubbles.

Complementary exams

Chest x-ray. In chronic bronchial patients without complications it can be completely normal. Only reinforcement of the normal shadows of the lung, hilum and weft may appear.

Syndromogenesis and etiology

1. Chronic bronchitis, per se, is caused by bronchial hypersecretion (hypertrophy of the mucous glands), metaplasia and inflammation, which can be defined in clinical terms by the presence of cough and expectoration daily, or almost daily, for three months a year, at least for two consecutive years, in the absence of other diseases that could explain these symptoms.

Tomas.

It is a process associated with exposure to bronchial irritants (tobacco, environmental pollution, occupational exposure) and respiratory infection. It has two great ends of the clinical spectrum:

a) Simple chronic bronchitis, without chronic airflow limitation.

b) Chronic brongchitis with chronic airflow limitation.

2. Symptomatic chronic bronchitis, accompanying or secondary to respiratory and non-respiratory diseases.

rias, as they are:

a) Respiratory causes: pulmonary fibrosis, pneumoconiosis, bronchiectasis, tuberculosis, lung abscess, bronchial stenosis, bronchopulmonary

monars.

b) Non-respiratory causes: some heart diseases, nephropathies, diastatic diseases, such as diabetes, obesity and mucoviscidosis.

Note: In practice, chronic bronchitis is observed closely associated with emphysema and is included under the term "chronic obstructive pulmonary disease" (COPD).

BRON UIAL OBSTRUCTIVE SYNDROME

Concept

It is characterized, as its name indicates, by a total or partial obstruction of the bronchi, which can appear suddenly or more slowly, depending on the cause that produced it.

Syndromography or positive diagnosis

Clinical picture

If it is an acute obstruction, such as when a foreign body is aspirated, the symptoms are loud with paroxysmal cough, dyspnea and sometimes cyanosis or exaggerated redness of the facies.

If the obstruction is slow, the picture is initially less striking; A not so persistent cough appears, but it can lead to emphysema or localized atelectasis, depending on whether the obstruction is partial or total.

The physical examination will vary according to the cause of the obstruction, the way it is installed and its consequences.

Localized examination:

Inspection: decreased respiratory mobility in the area.

Palpation: localized decrease in vocal vibrations.

Percussion: hypersonority of the affected area.

Auscultation: decreased vesicular murmur and localized dry rales.

Complementary exams

The chest x-ray sometimes shows the cause of the obstruction (radiopaque foreign bodies, tumors, lymphadenopathy or other cause), or the indirect signs of the obstruction such as an area of greater radiolucency located at the level of the affected bronchus. In chest x-rays performed during forced expiration, air trapping is confirmed, and mediastinal deviation towards the healthy side can be observed.

BRON UIAL ASTHMA SYNDROME FIG.

Concept

This condition is expressed clinically by a bronchial obstructive syndrome with special characteristics, since in bronchial asthma, in addition to airway obstruction, reversible spontaneously or with treatment, there is bronchial inflammation and their hyperreactivity to various stimuli.

Syndromography or positive diagnosis

clinical picture

It is characterized by sudden expiratory bradypnea unrelated to effort, sometimes accompanied by an annoying, dry cough that forces the patient to adopt a sitting position, which allows them to expand the rib cage as much as possible; In the final phase of the crisis, adherent, pearly mucous sputum may be expelled, its appearance coinciding with the patient's relief.

Physical examination:

Inspection: short inspiration and long expiration (expiratory bradypnea), orthopnea and chest in forced inspiration with presence of indrawing.
Palpation: decreased amplitude of the chest with palpable snoring and normal or decreased vocal vibrations.

Percussion: normal, slight hypersonority with decreased cardiac dullness due to distension of the lung reeds.

Auscultation: widespread hoarse and wheezing rales, sometimes audible at a distance. Subcrepitant rales may appear at the end of the crisis.

Complementary exams

In the posteroanterior chest x-ray, an increase in the transparency of the entire lung field can be seen during the crisis with reinforcement of the linear lines that correspond to bronchi and vessels. The diaphragm is very lowered.

In the lateral film, the globular appearance of the thorax and the flattening of the diaphragmatic domes are well observed.

Syndromogenesis and etiology

- Atopic or allergic asthma (extrinsic).
- Non-atopic non-reaginic asthma (related to respiratory infections).
- Aspirin sensitive asthma.
- Occupational asthma.
- Allergic bronchopulmonary aspergillosis.
- Others.

BRON SYNDROME UIECT SIC0 FIG.

Concept

They are bronchial dilations of different types that are usually accompanied by an infectious process.

It can be defined as the permanent abnormal dilation of one or more bronchi, due to destruction of the elastic and muscular components of the bronchial wall.

According to the appearance of the bronchi on bronchography, the terms saccular (cystic) bronchiectasis are used, mainly in the large proximal bronchi, cylindrical (fusiform) bronchiectasis, which affects the sixth to tenth generation bronchi, and varicose bronchiectasis, intermediate between the previous two.

Syndromography or positive diagnosis

Clinical picture

They can cause a cough with expectoration that is abundant in the mornings when you get up and that is purulent and sometimes foul-smelling. This type of expectoration does not occur in so-called dry hemoptoic bronchiectasis, which frequently occurs in the upper lobes.

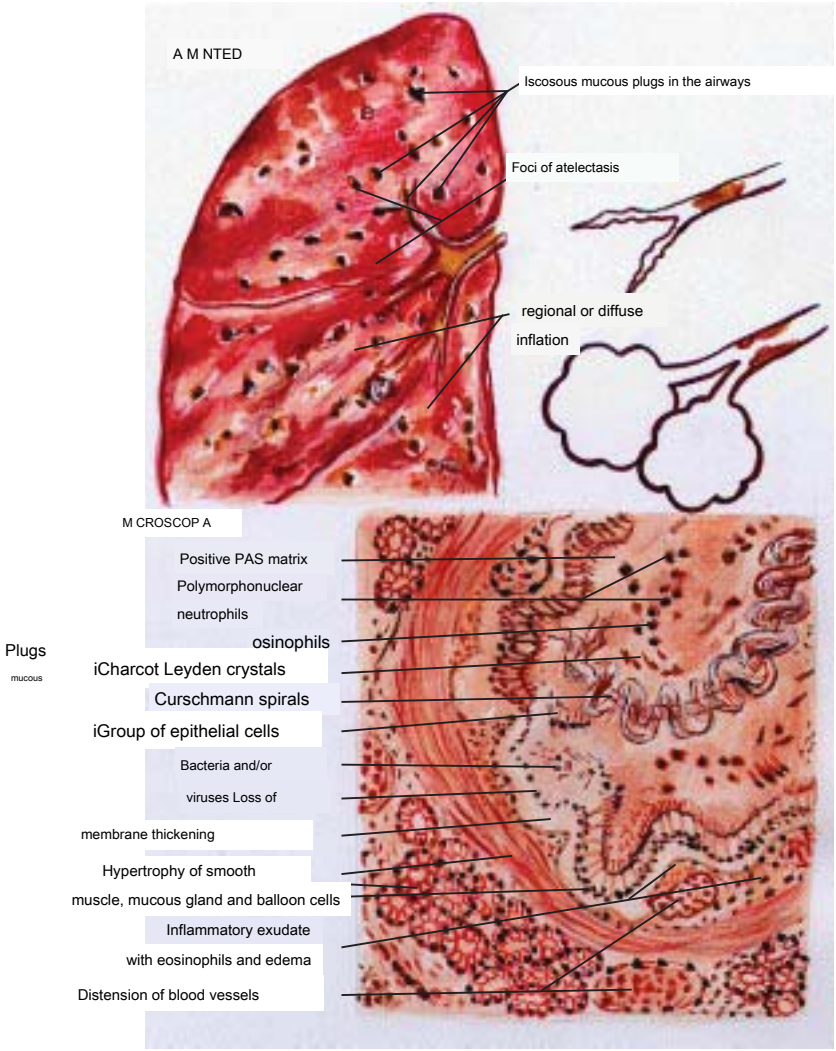


Fig. 36.1 Bronchial asthma.

Like any chronic bronchopneumopathy that is accompanied by sustained anoxia, it can lead to clubbing of the fingers.

The physical examination will vary according to the location of the bronchiectasis, its type (sacciform, tubular), the degree of infection of the bronchus or the surrounding parenchyma (peribronchiectasis pneumonia) and the degree of its refilling by secretions.

Complementary exams

In the simple chest x-ray, a reinforcement of the bronchovascular network can be seen, sometimes showing rounded images in the form of transverse rings.

superimposed parents, which translate dilated bronchi captured transversely. The diagnosis is confirmed with bronchography, in which a contrast material is used that shapes the bronchial lumen and allows its dilations to be observed.

Syndromogenesis or physiopathology

The most important cause seems to be necrotizing inflammation, usually of infectious origin, associated or not with genetic factors, such as the so-called "immotile cilia syndrome", which includes Kartagener syndrome. Recently this criterion has been expanded with ciliary dyskinesia that affects other patients.

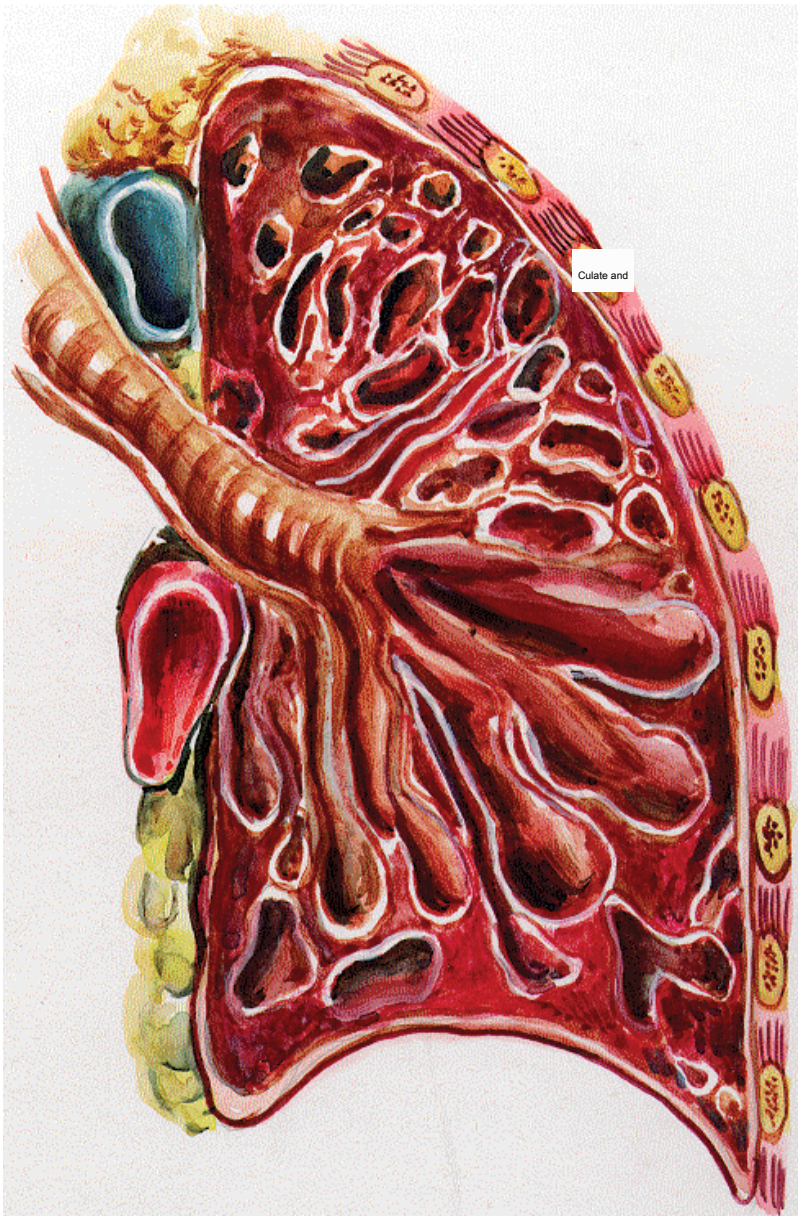


Fig. 36.2 Advanced saccular and cylindrical bronchiectasis.

tiology

It has been noted in pathophysiology, almost always with an infectious basis.

PULMONARY SYNDROMES

The lung parenchyma can be injured by several diseases, which usually produce various forms of bronchial and interstitial alteration together with alveolar pathology.

In this group we include those syndromes in which the parenchymal component prevails.

We will study:

- Emphysematous syndrome.
- Inflammatory condensation syndrome.
- Atelectatic condensation syndrome.
- Tumor condensation syndrome.

Cavitary syndrome

EMPHYSEMATOUS SYNDROME FIG.

We are going to refer only to generalized emphysema of the obstructive type in which there is a dilation of the pulmonary alveoli with a decrease in pulmonary elasticity secondary to bronchial obstruction.

Syndromogenesis or pathophysiology

A transient acute lung inflation may occur in bronchial asthma, but when the obstruction is chronic, as in recurrent bronchitis, the alveoli break their walls and fuse with each other, forming thin-walled bullae with loss of elastic tissue and trapped air content. Lung perfusion is also compromised due to arteriolocapillary mutilations.

All of the above gives rise to various types of respiratory failure. In severe cases there is pulmonary hypertension that affects the right heart (cor pulmonale).

Generalized emphysema can be complicated by interstitial emphysema, mediastinal emphysema, and pneumothorax.

Syndromography or positive diagnosis

Clinical picture

The main symptom is dyspnea caused by the slightest effort, or, what is more common, sustained dyspnea. The patient may experience "air hunger" that he tries to compensate by adopting the special position with the elbows resting on the knees and the shoulders elevated to take advantage of all the thoracic capacity; for this purpose he uses all the accessory muscles of breathing.

Dyspnea may be wheezing, as in bronchial asthma. Respiratory infections make it worse, fever, cough and expectoration may appear.

Physical examination:

Inspection: barrel chest already described.

Palpation: decreased expansive movements; The patient uses the abdominal muscles a lot for breathing. Vocal vibrations are decreased.

Percussion: pulmonary hypersonority. Reduction of cardiac dullness and decrease in lung bases.

Auscultation: decreased vesicular murmur with prolonged expiration. Wheezing and even wet rales may appear when an infectious condition is associated.

Complementary exams

The posteroanterior chest x-ray shows an image similar to that of bronchial asthma, but with high magnification of linear images that reflect a fibrous component. The great alveolar distension determines a decrease in the vascular network. There is an increase in the intercostal spaces, the ribs appear horizontal and the diaphragm descended with an increase in the vertical diameter.

In fluoroscopy, a decrease in diaphragmatic incursions is observed and the diaphragms appear almost flat with effacement of the costophrenic sinuses.

INFLAMMATORY CONDENSATION SYNDROME

This syndrome includes inflammatory pneumopathies of the pneumonic type, with the lobar (lobular) type being the most florid in its clinical manifestations.

In practice two syndromes must be considered:

I. Lobar (lobar) O pneumonic inflammatory condensation syndrome.

II. Lobular, multifocal or bronchopneumonic inflammatory condensation syndrome.

Lobar or pneumonic inflammatory condensation syndrome

Syndromogenesis or physiopathology

In these processes, the alveoli fill with an exudative material based on fibrin and leukocytes, as a result of lung inflammation. The process may be limited to one lobe (pneumonia) or be diffuse, as occurs with bronchopneumonia in which several parts of the lungs are affected, with the addition of a bronchial component.
Sometimes what becomes inflamed is the pulmonary interstitium, as occurs in viral pneumonitis.

Syndromography or positive diagnosis

Clinical picture

We are referring mainly to lobar (lobar) pneumonia. The symptoms correspond to those of an infectious process of the respiratory system.

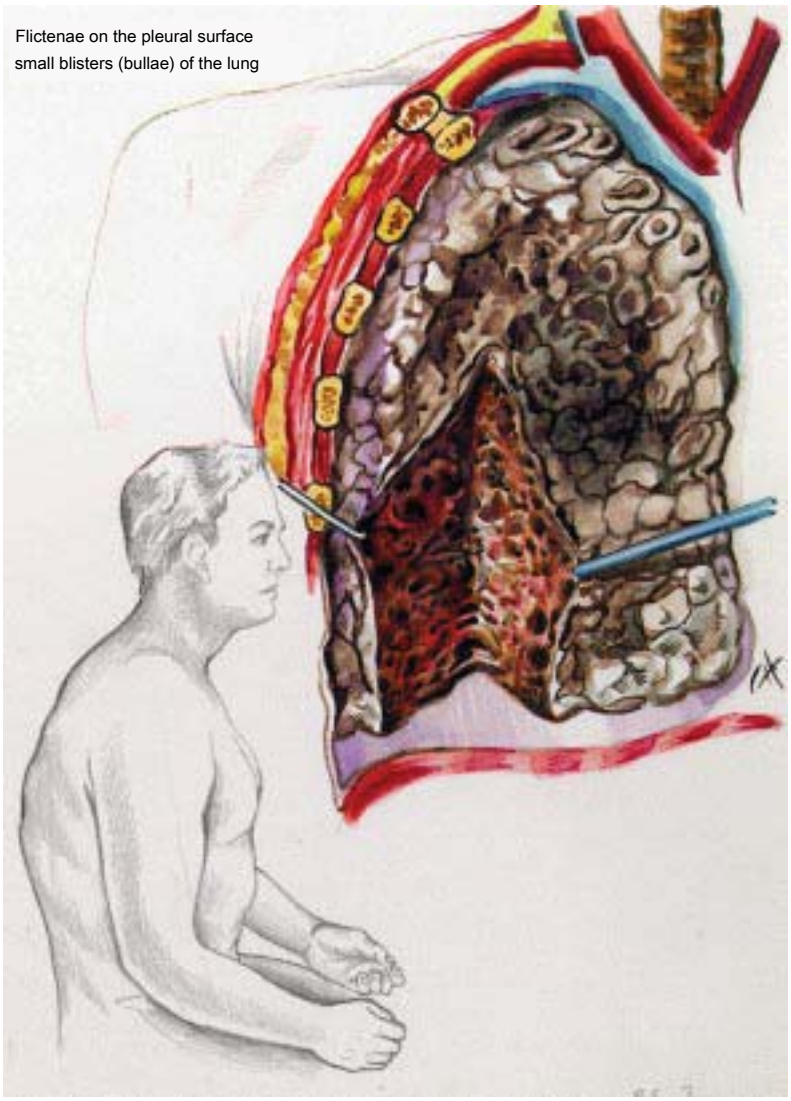


Fig. 36.3 Emphysema.

The patient may present chills, side pain, fever, dry cough at first and then productive with purulent, rusty expectoration that can sometimes become hemoptoic.

Often the symptoms are not so obvious.

The physical signs will vary according to the extent and depth of the pneumonic focus, since if the pneumonic condensation is far from the lung surface the physical examination may be normal and if it is of little extent, even if it is close to the surface, we may find a slight increase in vocal vibrations, a slight dullness and wet rales.

If the pneumonic condensation is extensive (fig. 36.4) and occupies an entire lung lobe, we will find:

Inspection: decreased or delayed breathing on the affected side.

Palpation: increased vocal vibrations.

Percussion: dullness.

Auscultation: absence of vesicular murmur, tubal murmur, crackles and subcrackles, increased bronchophony and voiceless

Complementary exams

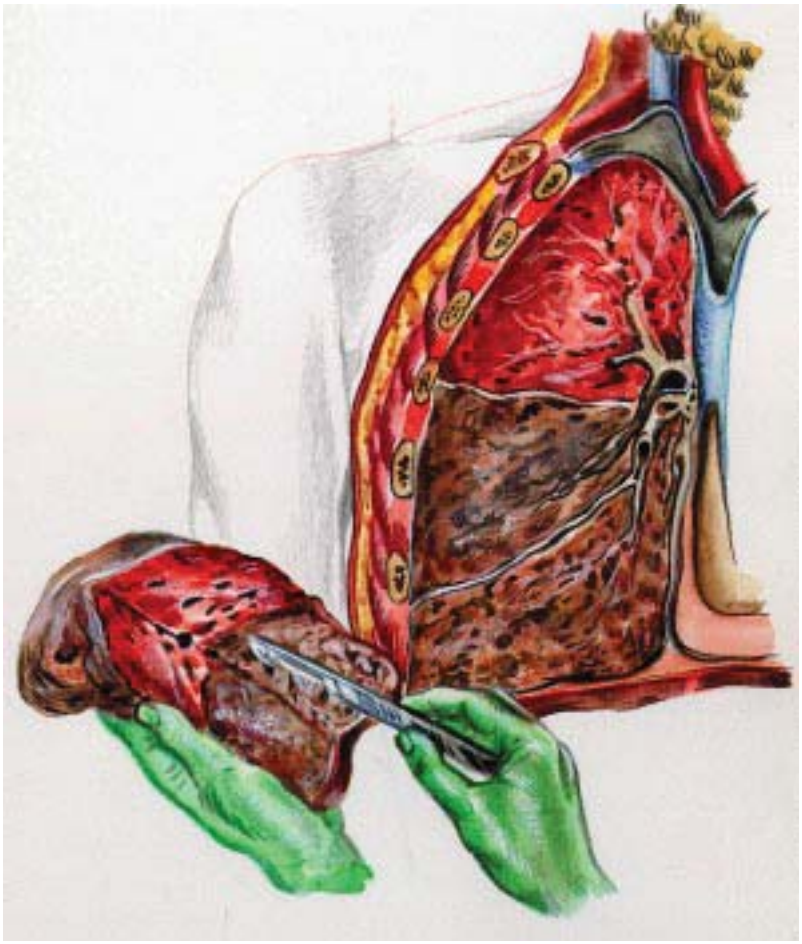
Chest x-ray. At the beginning, a diffuse veil opacity appears, which quickly increases in density and extends, sometimes adopting a triangular shape with a base towards the periphery. The consolidation of the process is usually reflected by a dense, homogeneous, well-defined shadow that occupies one or more lung lobes. The lateral plate allows us to more accurately locate the location of the process and its shape in which the apex directed towards the pulmonary hilum can be specified.

tiology

- Pneumococcus (Streptococcus pneumoniae): 90-95%. The most common are types 1, 3, 7 and 2.

Type 3 produces a very virulent form and lobar pneumonia.

Fig. 36.4 Pneumonic condensation.



- Less frequent:

Klebsiella pneumoniae.

Staphylococcus.

Streptococci.

Haemophilus influenzae.

Some gram-negative microorganisms such as: Pseudomonas and

Multifocal or bronchopneumonic lobular inflammatory condensation syndrome

Syndromogenesis or pathophysiology

A focal, actually multifocal, consolidation of the lung occurs. In infectious processes, the foci are areas of suppurative consolidation. Infection of the parenchyma usually originates from extension of previous bronchiitis or bronchiolitis. Lobular consolidation presents a patchy distribution, which can affect a single lobe, but is generally multilobar and frequently bilateral and basal, due to the proclivity of secretions to accumulate in the lower lobes, due to the action of gravity. There are areas of alveolar condensation (pneumonia) and peribronchiolar involvement, with foci of peribronchiolar inflammation.

(bronchopneumonia). The starting point may be aspiration of nasopharyngeal or gastric secretions, direct inoculation of microorganisms by air and hematogenous dissemination.

Syndromography or positive diagnosis

Clinical picture

a) Background.

Elderly, bedridden, malnourished patients, with previous respiratory tract infections, or with aspiration of gastric contents (bronchoaspirations).

b) General symptoms and signs.

Dyspnea, which may vary depending on respiratory compromise; from light to intense. Frequent cough. Mucopurulent expectoration. Fever, usually between 38 and 39.5 °C. Take the general condition. Accelerated pulse.

c) Respiratory examination.

Inspection: global decrease in bilateral or unilateral thoracic expansibility, depending on the degree of involvement. Polypnea.

Palpation: increased vocal vibrations.

Percussion: subdullness, if there is confluence of foci (more evident in the pseudolobar form).

There may be tympanic resonance if the foci are disseminated in the middle of lung tissue that replaces the function of the diseased areas.
Auscultation: bronchovesicular respiration or low intensity, variable and mobile tubal murmur. Scattered foci of crackles and, above all, fine subcrackles.
Bronchophonia. Hoarse and whistling rales, depending on bronchial participation.

Complementary exams

Chest x-ray. Poorly limited opacities, of variable size, in both lung fields.

tiology

infectious causes

Out-of-hospital or outpatient bronchopneumonia.
The main germs are: Streptococcus pneumoniae (pneumococcus), which generally expresses itself as pneumonia, with a lobar inflammatory condensation syndrome, staphylococci, Mycoplasma pneumoniae, Legionella pneumophila, Haemophilus influenzae, Pseudomonas aeruginosa, gram-negative bacilli, viruses, etc.

In-hospital or nosocomial bronchopneumonia. The main germs are gram-negative bacilli due to enterobacteria, such as Klebsiella pneumoniae, Enterobacter aerogenes, Escherichia coli, and non-enterobacteria, such as Pseudomonas aeruginosa. They can also be produced by gram-positive cocci such as Staphylococcus aureus and pneumoniae, and to a lesser extent, fungi and Legionella pneumophila.

Non-infectious causes of lung condensation

- Cardiogenic pulmonary edema and respiratory distress in adults.
- Pulmonary infarctions.
- Neoplasms.
- Extrinsic allergic alveolitis.
-Vasculitis and eosinophilic syndrome.
- Chemical pneumonitis.

ATELECT SICA CONDENSATION SYNDROME FIG.

In this case, the alveoli fold on themselves with the disappearance of the gaseous content, due to processes that obstruct the airways, interfering with the entry of air and thereby allowing the alveolar content to pass into the blood; This causes pulmonary atelectasis syndrome.

Syndromogenesis or pathophysiology

Although the parenchymatous component is the one that draws attention in this picture, and therefore, occupies its

Among the pulmonary syndromes, the production mechanism is located fundamentally at the level of the bronchus, whose obstruction can be of intrinsic or extrinsic cause. Of intrinsic cause, we can mention airway tumors, stagnation of exudates and foreign bodies, among others, and of extrinsic cause, compression of the bronchial lumen due to tumor masses, lymphadenopathy or another cause.

Once the bronchus is obstructed, the alveoli lose their gas content as gases pass into the blood, their walls fold and the capillaries become engorged; If the obstruction does not disappear, allowing the affected area to be ventilated again, fibrosis is then organized with total loss of the characteristics of the organ (atelectatic condensation).

Syndromography or positive diagnosis

Clinical picture

The symptomatic picture varies with the extent of the process. If the obstruction affects a main bronchus, the atelectasis will be massive; if a median bronchus, it will be partial or lobar; but if they are small caliber bronchi, segmental atelectasis will be constituted. The latter usually lack clinical manifestations, but the massive and lobar manifestations, especially if they are sudden in onset, are accompanied by dyspnea, side tip and fever.

Physical examination:

Inspection: decreased respiratory expansion. In massive atelectasis there may be retraction of the hemithorax, indrawing and reduction of the intercostal spaces.
Palpation: decreased respiratory expansion.
Vocal vibrations are diminished or abolished.
Percussion: dullness. In large-scale atelectasis, the deviation of the dullness of the mediastinum towards the affected side can be seen.
Auscultation: vesicular murmur abolished with respiratory silence and bronchophony diminished or abolished.
No voice auscultation.

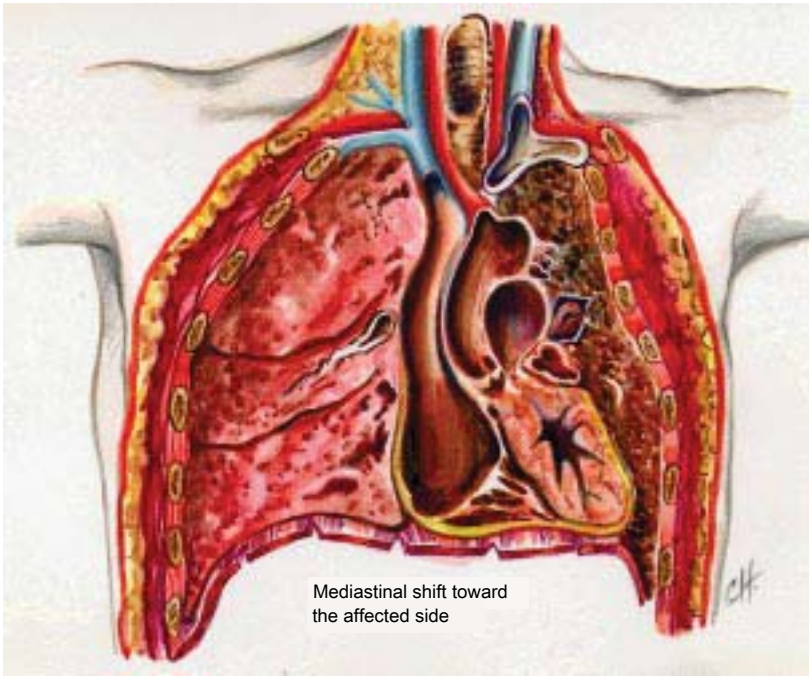
Complementary examinations

Chest x-ray. In partial atelectasis, there is complete opacity of the affected lobe and greater transparency of the remaining lobes due to vicariant function.

The bronchographic examination demonstrates the level of the obstruction and the lack of bronchial filling in the compromised area.

In massive atelectasis, the opacity of the hemithorax is total and homogeneous; The retraction of the hemithorax and the intercostal spaces is seen, increasing the obliquity of the ribs.

Fig. 36.5 Atelectatic condensation.



Blood plug occluding the bronchus (bronchoscopic view)

The elastic structures of the tőrax tend to compensate for the increase in pleural void and the deviation of the mediastinum towards the diseased side is observed, which allows the transverse processes of the dorsal column to be distinguished on the healthy side. The area of tracheal transparency also deviates towards the side of atelectasis, as does the corresponding hemidiaphragm, which rises attracted by the process.

TUMOR CONDENSATION SYNDROME

We will not insist on this syndrome, since its manifestations depend on:

1. Its nature.
2. Its location.
3. Its volume.
4. The degree of invasion.
5. The presence of metastases.
6. Secondary infection.
7. The modifications they make to the various structures of the respiratory system.

That is why, in addition to a general syndrome (asthenia, anorexia and weight loss), lung cancer may present symptoms and signs of the respiratory system.

It occurs in the form of a bronchial syndrome, a parenchymal syndrome or a pleural syndrome, so varied in their clinical forms that they range from an apparent chronic bronchitis to atelectasis or a pleural effusion.

Syndromography or positive diagnosis

Clinical picture

It becomes manifest in invasive bronchopulmonary cancer, in which there is an absence of bronchial and alveolar

Generally there is asthenia, anorexia, weight loss and persistent pain, such as subacute side pain. The pain may be localized in specific areas or may present with intense, continuous pain, with paroxysms, such as intercostal neuralgia of the brachial plexus.

Physical examination:

General inspection. Hypertrophic pneumatic osteoarthropathy may occur. Claude-Bernard-Horner syndrome is observed in Pancoast-Tobías tumor, in tumors of the pulmonary or pleural apex and that injure or invade the cervical sympathetic stellate ganglion.

Respiratory exam:

Inspection: there may be hemithoracic retraction (ipsilateral Bosco contortion) and decreased expansion of the affected hemithorax.

Palpation: vocal vibrations abolished.

Percussion: dullness.

Auscultation: absence of vesicular murmur. Other signs corresponding to cavitation lesions or associated pleural effusion can be added.

Complementary exams

As early diagnosis is of extraordinary importance (which at the same time is difficult), we have to rely on simple and contrasted radiographs and endoscopic, cytological or other studies to be able to reach its diagnosis.

In the simple chest film, opacities of very variable appearance may be present: rounded, infiltrative, atelectatic, and even cavitary, when the tumor has undergone necrosis processes inside. The borders may be defined in benign tumors, but not very precise due to infiltration, in very malignant tumors.

CAVITARY SYNDROME

This syndrome can be observed in cavities more than 3 cm in diameter, surrounded by condensed parenchyma and communicating with a patent bronchus. When the caverns do not present pericavitary condensation, or are very deep as in the parahilar region, no cavity signs are appreciated.

Syndromography or positive diagnosis

Respiratory exam:

Inspection: localized retraction of the chest, inconsistent. Decreased chest expansion on the affected side.

Palpation: increased vocal vibrations.

Percussion: dullness caused by condensation. Sometimes, tympanic resonance or tympanicism. Other times, metallic tympany (large caverns) or a "crashing pot" noise.

Auscultation: bronchial respiration or cavernous murmur, more rarely amphora (large cavities), bronchophony and pectoriloquy or amphorophonia.

The chest x-ray reveals the cavern, sometimes with an air-fluid level, if they are large.

tiology

-Tuberculous caverns (most common).

- Bronguiectasias.

- Abscesses.

-Excavated tumors.

-Open hydatid cysts.

PLEURAL SYNDROMES

There are two main pleural syndromes:

- Liquid interposition syndrome or pleural effusion.

- Air interposition syndrome or pneumothorax.

In both cases, the virtual pleural cavity becomes a real cavity, with a certain content, which in the first case will be liquid: serofibrinous, hemorrhagic or purulent pleurisy, and in the second it will be air that will penetrate through a perforation of the parenchyma or the chest wall. Hydrothorax has the same semiogenesis as edema.

The physical signs that translate these conditions are multiple, but characteristic and allow the diagnosis with quite certainty.

LI UIDA INTERPOSITION SYNDROME 0 PLEURAL EFFICIENCY

Any inflammatory, irritative or mechanical condition that affects the pleurae or compromises return circulation is capable of causing a pleural effusion. It should be noted that effusions in adults, less than 400 mL, and in children, 120 mL, are not manifested by any clinical sign.

Syndromogenesis or pathophysiology

The accumulation of fluid in the pleural space is a consequence of a disorder in the normal balance between transudation and reabsorption, which can be caused by various processes. In this way, the increase in microvascular hydrostatic pressure, the alteration and obstruction of lymphatic drainage, the reduction of osmotic pressure, the contribution of fluid from the peritoneal space, through small transdiaphragmatic communications or the increase in the permeability of the microcirculation, due to inflammation or tumor invasion of the pleura, can cause a pleural effusion.

The physical conditions created in the thoracic cavity by a body that is a poor conductor of sound, such as liquid bodies, is the cause that explains the phenomena that occur at that level. For this reason, the amount of the spill creates different conditions, both in the symptomatic and signological aspects.

Syndromography or positive diagnosis

Clinical picture

The clinical picture usually begins with the so-called "dry pleurisy" with dull pain, shallow breathing, and a non-productive and annoying cough. Chills are not uncommon

and general feverish malaise or high fever. Once the effusion is established, the condition acquires greater intensity, presenting a side tip sometimes of an abdominal type, breathing becomes dyspneic depending on the abundance of fluid and the presence or absence of parenchymal lesions, the cough persists dry but annoying and continuous. If the fluid continues to increase, the condition becomes extraordinarily worse, with the primary symptom being intense dyspnea with marked cyanosis and anoxia.

Physical signs vary with the amount of effusion:

a) In small volume spills (500-1,000 mL) (fig. 36.6).

Inspection: normal.

Palpation: decreased respiratory expansion.

Vocal vibrations decreased in the basal posterior plane, but not in the axillary and upper posterior plane.

Percussion: dullness behind that does not exceed the posterior axillary line.

Auscultation: decrease in vesicular murmur and bronchophony in the area of dullness.

b) In medium volume spills, more than 1,500 mL.

Inspection: discreet vaulting of the chest. Decreased thoracic expansion.

Palpation: distension of the hemithorax and decreased expansion are

Vocal vibrations abolished; If they are seen, it means that there is joint condensation of the parenchyma below the effusion. Above the effusion there is increased vocal vibrations.

Percussion: dullness, resistance to the percussing finger.

The dullness forms a parabola, which goes from the spine behind, to the sternum in front, with its highest point at the level of the mid-axillary line (Damoiseau curve).

Matte spine to percussion.

On the opposite side to the effusion there is a triangular area of dullness in the inner part of the base that corresponds to the so-called Grocco triangle.

On the side of the effusion, at its upper limit, a triangular zone of submatidity is formed, which has as its internal limit the vertebral column and as its external limit the ascending curve of the effusion and which is called Garland's triangle.

Above the effusion there is hypersonority or tympany (scodism) due to the replacement function of the parenchyma not collapsed by the liquid.

If the effusion is on the left side, the sound of the stomach may disappear (Traube's semilunar space).

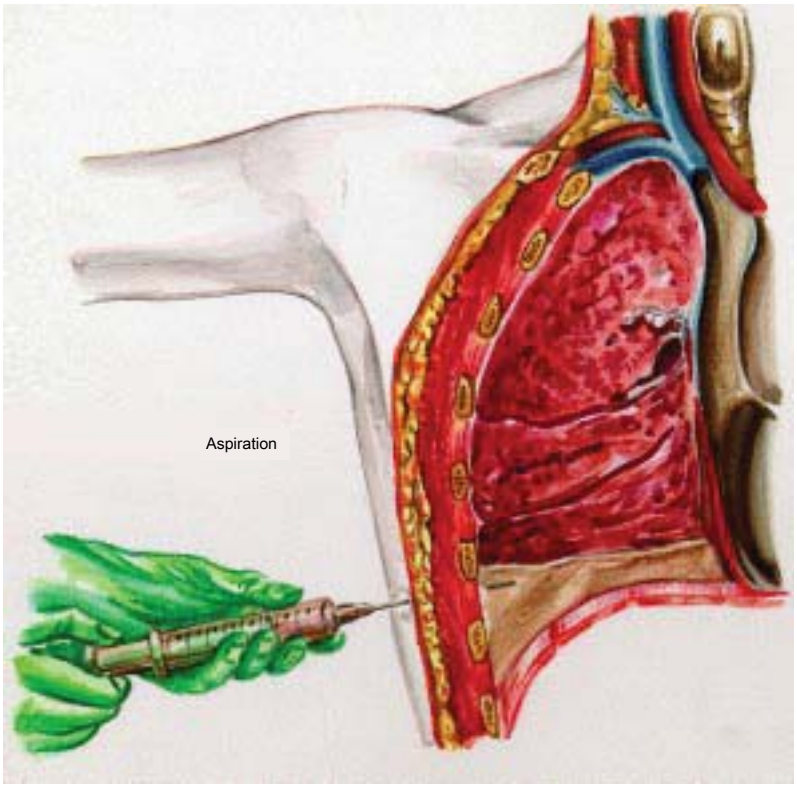


Fig. 36.6 Pleural effusion.

Auscultation: at the level of the effusion there is a very marked decrease or abolition of the vesicular murmur. There may be bronchial respiration of variable character.

Pleuritic murmur, soft, veiled, expiratory.

When auscultating the voice: voiceless pectoriloquy. At the upper limit of the spill: egophony.

Above the effusion, the vesicular murmur is intense and sometimes there are pleural rubs and crackles, due to added pulmonary congestion.

Pitres currency sign.

c) In large volume spills, more than 3,000 mL.

Inspection: vaulting of the chest. Distended intercostal spaces. Immobility of the hemithorax.

Palpation: absence of expansive movement. Vocal vibrations abolished.

Percussion: dullness throughout the height of the hemithorax.

The deviation of the dullness of the mediastinum towards the side opposite to the effusion is also verified.

Auscultation: Abolition of vesicular murmur. Auscultation of the voice negative or absent. Currency sign.

Complementary exams

Chest x-ray. In small effusions, an opacity of the base appears that erases the costophrenic and cardiophrenic angles, with a horizontal appearance. As the liquid increases, the opacity is greater and in medium-volume spills a curve with greater external height and imprecise limits corresponding to the classic Damoiseau curve is observed. In large effusions, the opacity extends to the entire hemithorax, observing how the mediastinum shifts markedly toward the healthy side, the intercostal spaces distended, and the corresponding diaphragms descended.

In addition to the posteroanterior (PA) chest x-ray, lateral and in Pancoast position, add:

Ultrasound: it is capable of detecting small spills.

Thoracentesis or thoracentesis (pleural puncture).

Study of pleural fluid.

A. Thoracentesis or thoracentesis (pleural puncture).

It allows obtaining the fluid and performing bacteriological and cytological tests to determine the etiology of the syndrome.

drome.

Thoracentesis, thoracentesis or pleural puncture is a very useful diagnostic technique that, in effusions of considerable volumes and in pneumothorax, also meets therapeutic objectives.

The approach varies depending on the extraction, whether air (pneumothorax) or liquid. In the first case, it is done in the anterior plane, at the level of the second intercostal space, in the midclavicular line.

Technique:

To extract the liquid, after clinical, radiological or even ultrasound verification, the area to be manipulated is sterilized. An intercostal space is chosen at the level of the posterior axillary line, below the second and up to the eighth intercostal space (only in special circumstances is it done in the ninth and up to the tenth, but never below this). It is properly sterilized and anesthetized with plane infiltration, always introducing the needle above the upper costal edge of the lower rib of the intercostal space in question.

After plane anesthesia, the presence of liquid is checked with a needle and replaced with a trocar designed for this and with a syringe with a control mechanism (ideally a three-step key) the extraction is carried out, according to the diagnostic or therapeutic objectives or both.

It is not advisable to extract more than one liter initially, due to the danger of pulmonary edema.

The extracted liquid, in addition to direct observation of its appearance, color, transparency, viscosity (we can check it with gloved fingers), presence of blood, etc., is sent to the corresponding laboratories for cytochemical, microbiological and cytological analysis.

With these elements we can appreciate if we are in the presence of a transudate, an exudate, a chylous effusion, a purulent effusion, a serohemaic effusion or a hemothorax.

B. Study of pleural fluid.

It is necessary to make some definitions about the main types of pleural fluids.

Transudate:

It has a protein content of less than 3 g/dL with normal serum protein concentrations.
Pleural fluid lactic dehydrogenase (LDH) less than 200 IU. The pleural fluid LDH/serum LDH ratio is less than 0.6. Rivalta test negative (will be described later).

Exudate:

The protein content is greater than 3 g/dL with a 'pleural fluid protein/serum protein' ratio greater than 0.5. The pleural fluid LDH is greater than 200 IU. The pleural fluid LDH/serum LDH ratio is greater than 0.6. Rivalta test positive. serohematic.

Empyema:

It is purulent pleural effusion. It can be loculated.

The pH is low. Leukocytes with polymorphonuclear or lymphocytic predominance. Germs can be identified by direct microbiological examination with Gram stain.

Hemothorax:

It is called this when, unlike serohematic effusion, the amount of blood in the pleural space is significant. The hematocrit of the fluid is usually more than 50% of that of blood.

Lipid spills (chylothorax and pseudochylothorax):

These effusions are characterized by high concentrations of lipids, as well as a milky or cloudy appearance. It is called gulothorax when chyle penetrates into the pleural space, from the thoracic duct. It is called pseudochylothorax, when there is an accumulation of large amounts of cholesterol or lecithin-globulin complexes, causing a pleural effusion.

Chylothorax has an increase in lipid content at the expense of chylomicrons (their presence confirms the

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Amber color and not very viscous More intense yellow color, reminiscent of urine Bloody appearance	hydrothorax Inflammatory pleuritis Bronchopulmonary cancer Tuberculous pleurisy
Cloudy, homogeneous, greenish yellow, very viscous Putrid, fetid, fecaloid odor	Pneumonic empyema Anaerobic germs

e ci ic del| you

Cellular	
Polymorphonuclear	Acute bacterial inflammatory processes Viral processes.
lymphocytes or monocytes	Tuberculosis. Malignant conditions. Mycoses. Other chronic inflammatory processes
Eosinophils (more than 10)	Loeffler syndrome. Panarteritis nodosa. Odgkin's disease Malignant processes
Atypical cells	
uimic (others, in addition to those already mentioned in the text)	
Calcium	Low figures in transudates
Amylase	High in acute pancreatitis
Glucose	In transudates: values similar to those of blood Its decline (less than 40-50 mg dL) is typical of inflammatory and rheumatic effusions. Its absence or very low presence, in empyemas. Positive in exudates
Rivalta test	
Intracellular cytochemistry	
DL arrest	Sign of malignancy
Acid phosphatase in intraprotoplasmic vacuoles	Pleural metastasis from prostate cancer

diagnosis), increased triglycerides and normal cholesterol. Sudan stain III positive.

In pseudochylothorax the fluid is negative for Sudan III and a high cholesterol content is present.

Rivalta test:

In protein-rich exudates, it is positive. Two drops of acetic acid are poured into 100 mL of water contained in a test tube. A drop of the spill is added to this solution with a pipette. If it is an exudate, when the drop falls it forms a white cloud on its way to the bottom that has been compared, due to its appearance, to cigarette smoke. If it is a transudate, this cloud is very faint or barely perceptible.

tiology

A. Transudates.

1. By increased hydrostatic pressure:

Congestive heart failure.

Constrictive pericarditis.

Superior vena cava syndrome.

2. Due to decreased oncotic pressure, due to hypoalbuminemia:

Liver cirrhosis.

Nephrotic syndrome.

3. Associated with ascites (transdiaphragmatic communications)

Liver cirrhosis.

Peritoneal dialysis.

B. Exudates.

1. Infections:

Bacterial or viral pneumonia (parapneumonic effusion).

Tuberculosis.

2. Neoplasms:

Lung cancer.

Pleuropulmonary metastases.

Lymphomas. Leukemias.

Mesothelioma.

3. Intra-abdominal diseases:

Pancreatitis.

Subphrenic abscess.

4. Connective tissue diseases:

Rheumatoid arthritis.

Systemic lupus erythematosus.

5. Others:

Embolism and pulmonary infarction.

Reaction due to hypersensitivity to drugs.

Myxedema.

Idiopathic.

C. Empyema.

Bacterial pneumonia due to anaerobic germs, Staphylococcus aureus, Pseudomonas and Escherichia coli.

Chest trauma.

Thoracic surgery.

Mediastinitis.

Ruptured subphrenic abscess.

Lung abscess.

D. Chylothorax.

Chest trauma.

Lymphomas.

Post-surgical complication.

E. Pseudochylothorax.

Tuberculosis.

Rheumatoid arthritis.

Idiopathic.

F. Hemothorax (no serohematic effusion).

Penetrating or blunt trauma.

Iatrogen.

Metastatic pleural disease.

Complication of anticoagulant treatment.

AIR INTERPOSITION SYNDROME

OR PNEUMOTHORAX FIG.

Syndromogenesis or physiopathology

It consists of the interposition of a mass of gas (air) between the visceral and parietal pleura. Generally, when it occurs suddenly and in full health, it corresponds to the rupture of an emphysema bulla. In tuberculosis patients, it produces a serious condition, as it corresponds to the bursting of a cavern and gives rise to mixed or air-fluid syndromes that are invariably complicated by Koch bacillus empyema. Finally, they can be caused by trauma that perforates the thoracic cavity and allows air from outside to enter.

Syndromography or positive diagnosis

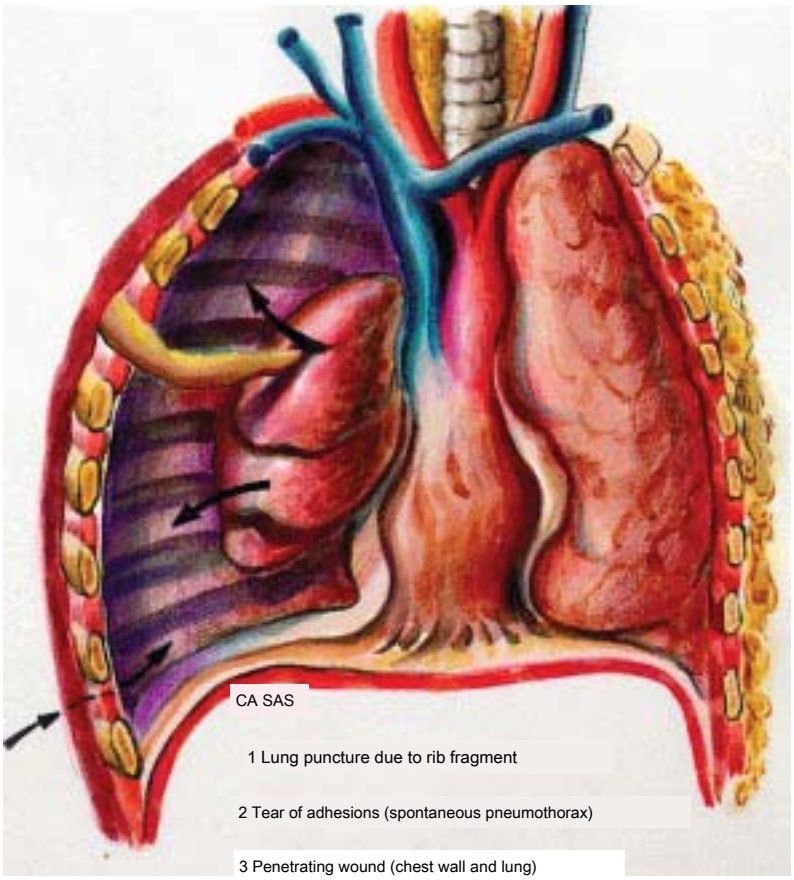
Clinical picture

It is usually dramatic, with side to side, intense distressing and progressive dyspnea, and cyanosis. When it develops slowly, it may only present with pain and slight dyspnea.

Inspection: vaulting and decreased expansion of the involved side.

Palpation: decreased thoracic expansion. Vocal vibrations abolished.

Fig. 36.7 Pneumothorax.


CA SAS
1 Lung puncture due to rib fragment
2 Tear of adhesions (spontaneous pneumothorax)
3 Penetrating wound (chest wall and lung)

Percussion: hypersonority, tympanism and if the pneumothorax is very extensive, metallic sound. In case of spillage, concomitant dullness at the base and loudness above with the indicated characteristics. Deviation of mediastinal dullness towards the opposite or healthy side.
Auscultation: decrease or absence of vesicular murmur.
Amphoric murmur if the perforation is of sufficient size. Decrease or abolition of bronchophony. Hippocratic succussion if there is effusion.

If the pneumothorax is partial, the symptoms will vary, and the aforementioned condition may even be completely absent.

Complementary exams

Chest x-ray. An increase in transparency is seen on the side corresponding to the pneumothorax, with the lung retracted towards the hilus, giving a characteristic stump shape. Generally, a discreet opaque level is observed towards the bases that erases the costophrenic and cardiophrenic angles and corresponds to the effusion that usually accompanies these processes. In partial pneumothorax, radiography is what generally establishes the diagnosis, specifying the free edge of the lung with the greater or lesser layer of gas that separates it from the chest wall. To detect a very small pneumothorax that is not evident in the usual chest and is strongly suspected, a posteroanterior view can be performed, in forced expiration.

tiology

1. Spontaneous pneumothorax:

a) Primary.

b) Secondary to an associated underlying lung disease:

Chronic obstructive pulmonary disease (most common)

.

Granulomatous diseases (tuberculosis, sarcoidosis).

Bronchogenic carcinoma.

Pulmonary fibrosis.

Catamenial pneumothorax (associated with menstruation).

2. Traumatic pneumothorax:

a) latrogen.

b) As a result of penetrating or closed injuries.

MEDIASTINAL OR MEDIASTINAL SYNDROME

Concept

It is the set of symptoms and signs with which conditions of diverse origin manifest themselves, which damage the

organs of the mediastinum and that may depend on compression. of the inflammation or destruction of these or the pleuropulmonary walls that surround it. Due to the diversity of clinical manifestations, they are often referred to as "mediastinal syndromes."

Syndromogenesis or pathophysiology

The symptoms and signs of mediastinal affections will be related, rather than to the cause, to the type of organ or organs injured and to their degree of affectation by the compressive, inflammatory, infiltrative or destructive factors that compromise the rich and varied content (heart, large venous and arterial vessels, duct and lymph nodes, trachea, bronchi, nerves, esophagus, thymus, etc.) of the space. mediastinal.

The symptomatic polymorphism of the syndrome or syndromes lies, on the one hand, in the quantity, variety and importance of the organs contained in a reduced space, which determines that due to contiguity the injury to one of them has repercussions on the neighbor. This is clinically translated by various symptoms and signs that make up a complex syndrome or even, as we already clarified, several syndromes. On the other hand, as many of the mediastinal organs perform transport functions, their injuries can have repercussions at a distance, that is, in regions far from the mediastinum.

Syndromography or positive diagnosis

Clinical picture

Symptoms:

At first they frequently appear in isolation.

Among the most frequent are: chest pain (usually neuralgic type, although it can be dark, due to bone erosion and even, although rarely, anginal type); dyspnea (permanent and increased by effort or paroxysmal); cough (which may be strong, at periodic intervals, thick or emetic); and dysphagia (which is usually progressive and continuous, of an organic type). Other symptoms may include: hemnoptysis, dysphonia, hiccups, headache, dizziness, drowsiness, ringing in the ears and epistaxis.

There may be a history of recurrent respiratory infection.

General physical examination:

Mediastinal facies (as a result of venous stasis, due to compression of the superior vena cava). It is characterized by edema in clavicle, or limited only to the face, neck or supraclavicular fossae; great engorgement of the jugular veins and cyanosis of the lips and extremities of the fingers of the face, neck and upper limbs.

Slavine edema (edema of the neck, head and upper limbs) .

Cyanosis (already described in facies).

Venous turgor of the vessels of the neck and upper limbs.

Venous network of thoracic or thoracoabdominal circulation.

Slavine edema, cyanosis of the hands and face, and collateral or complementary circulation form the classic triad of the "superior vena cava syndrome," within the great mediastinal syndrome.

Physical examination of the chest:

In addition to what was mentioned above, deformations can be found in the thorax, such as those caused by some aneurysms of the aortic valve, which invade and destroy the sternal manubrium. There may also be fistulous openings, abscesses that open to the outside.

Wenckebach's sign can also be detected, which consists of the lack of forward projection of the sternum during the inspiratory movement (more typical of pediatric practice).

By palpation of the suprasternal pit, the beating or expansion of an aortic aneurysm can be perceived, or the hardness of a tumor process in the upper mediastinum.

The classic sensitivity points of the phrenic and intercostal nerves are painful to acupressure.

Examination of the respiratory system:

Inspection: inspiratory dyspnea and indrawing; cornage, if compression of the

Palpation: vocal vibrations may be decreased, or even abolished, if there is bronchial obstruction.

Percussion: in the anterior plane dullness of the mediastinal region. In the presence of a thymoma, the thymic dullness is in the shape of a triangle with a superior base.

Skodism, tympanic resonance in the pulmonary apices.

In the posterior plane, submattitude can be seen in the interscapulovertebral regions, between the third and seventh dorsal vertebrae and the spinal edges of the scapulae.

Auscultation: a prolonged and even blowing expiration can be perceived, which can lead to bronchial respiration or tubal murmur, due to the propagation of sound through the mediastinal process. Depending on the degree of bronchial obstruction or compression: decrease or absence of vesicular murmur.

It has been described that auscultation of the loud voice and the whispered voice decrease in intensity and clarity, from the vertices to the bases (Martin du Magni sign).

Complementary exams

Chest x-ray. It remains a very useful diagnostic test. Provides information on the size,

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Clinical signs	Structure affected
Exophthalmia, conjunctival hemorrhages,	superior vena cava
macroglossia Collateral circulation, venous turgor and unilateral edema of the upper limb and corresponding side of the	Brachiocephalic trunk
neck Pulmonary congestion and hydrothorax right	Pulmonary veins Zygos major
Pleural effusion and chylous ascites	Thoracic duct
Murmur and thrill in the pulmonary focus	Pulmonary artery
Congestive epatomegaly, ascites, edema in lower limbs Porfour du Petit or Claude Bernard-orner syndromes	inferior vena cava Sympathetic
Bitonal voice, aphonia	Recurrent nerve
Bradycardia, tachycardia	Vagus

density and anatomical location of mediastinal masses. These can be recognized in the lateral and oblique views, having occupied the anterior clear space (between the cardiovascular shadow and the sternum) or the posterior one (between the vertebral column and the cardiovascular shadow). In the posteroanterior view, mediastinal masses are recognized by opacity of the hilum or lung fields, with mediastinal widening. Other characteristics can be seen, depending on whether they are vascular dilations, tumors, lymphadenopathy (for example, the polycyclic image in lymphomas).

Computed axial tomography (CAT). It is very valuable; It has displaced simple tomography and ultrasound in mediastinal conditions. Allows you to identify masses of different density. Your possibilities increase with the use of contrast materials.

Nuclear magnetic resonance (MRI). It has great value in the study of the posterior mediastinum.

Transthoracic fine needle aspiration biopsy (FNAB). It is useful in non-vascular mediastinal masses.

lares.

Other procedures may be: esophagram and esophagoscopy, bronchoscopy, mediastinoscopy with biopsy, angiography and scintigraphy.

tiology

Inflammatory causes

- Acute mediastinitis, especially suppurative.
- Chronic mediastinitis, due to tuberculosis, histoplasmosis, or of unknown cause that causes mediastinal fibrosis.

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Timoma	Previous
Germ cell tumor (Example teratoma)	Previous
Neurogenic tumor	Anterior, middle, posterior
Lipoma	Later
Congenital bronchogenic carcinoma Metastatic tumor	Former Half Middle and later Middle
c Diaphragmatic aortic aneurysm Achalasia	Anterior and posterior Posterior Later

RESPIRATORY FAILURE SYNDROME

Concept

Respiratory insufficiency syndromes are known as the set of symptoms, signs and analytical alterations of arterial gases, which appear in a patient in which the mechanism of oxygen uptake, CO elimination, or both, at the level of the pulmonary capillary bed, is disturbed by factors that alter the renewal of alveolar gas or the exchange between it and capillary blood, and which may correspond to multiple causes, which often coexist in the same patient.

It is important to exclude a right-to-left cardiac shunt and when hypercapnia exists, that this is not due to compensation for metabolic alkalosis.

Classification

There are various ways to classify respiratory failure. Depending on the intensity and form of onset, respiratory failure can be acute or chronic.
nica. Depending on how it manifests itself in the face of varying degrees of metabolic demands, it may be compensated or decompensated respiratory failure. It will be decompensated, if it appears in basal conditions. But, if it only occurs in the face of maximum levels of metabolic demands, it is considered compensated.

It can also be classified according to the values of the partial pressure of arterial gases (0, and CO,), into hypoxemic (pa0, low and paCO, normal or low) or hypercapnic-hypoxemic (pa0, low and paCO, high). This most recent pathophysiological-analytical classification has great practical interest for a better understanding of the patient and their therapeutic management. Finally, respiratory failure can be classified according to its etiopathogenesis, as we will see later.

Up to this point we can make some considerations that clarify and integrate some of these classifications:

Acute respiratory failure. Acute respiratory failure is that with a more or less sudden onset and potential reversibility, in a patient without a history of chronic respiratory failure. If it appears in the context of a previous chronic respiratory disorder with functional compromise, it is called acute chronic respiratory failure.

The acute state is also defined by the fact that renal compensation cannot occur and the pH decreases.

In acute respiratory failure, both in previously healthy and damaged lungs, there is a serious respiratory functional alteration, with great immediate risk to the patient's life.

Chronic respiratory failure. This is the case of a process of insidious onset, of chronic evolution, without potential complete reversibility. In this, renal compensation of hypercapnia occurs and the arterial pH remains close to normal, at the expense of an increase in serum bicarbonate.

The name stable chronic respiratory failure corresponds in the classification to compensated respiratory failure; and the decompensated one corresponds to that of chronic respiratory failure. only sharpened. The last two classifications will be treated in "Syndromogenesis".

Below we propose a classification of respiratory failure that includes all those that we have we have pointed out and that may be useful, from a didactic point of view:

I. According to intensity and form of appearance:

A. Acute.

B. Chronicle:

1. Stable (compensated).

2. Acute (decompensated).

II. According to the alterations in arterial gases:

A. Hypoxemic (paO, low and paCO, normal or low).

B. Hypercapnic-hypoxemic (paO, low and paCo, high).

III. According to its etiopathogenesis:

A. Hypoventilation (ventilatory failure):

1. Obstructive.

2. Restrictive:

a) Due to limitation of expansion:

Thoracic.

Pulmonary.

Diaphragmatic.

b) Due to neuromuscular defects.

c) Due to injury or depression of the respiratory center.

B. Imbalance in the ventilation/perfusion relationship.

C. Intrapulmonary shunt from right to left.

D. Disturbance or limitation of diffusion.

E. Mixed:

1. Ay B Association.

2. Association of B and C.

Syndromogenesis or pathophysiology

This syndrome can originate from alterations that compromise the anatomofunctionality of the respiratory system. It can occur in diseases themselves (of the airways and lung parenchyma) and also in those that affect the pulmonary vascularization, the chest wall, the respiratory muscles and the ventilation control system.

The four functional processes that make up respiration: ventilation, diffusion, irrigation and respiratory control, guarantee that normal values of partial pressure of oxygen and carbon dioxide in arterial blood (paO, and paCO,) are maintained. Therefore, the alteration of any of them, if it is of sufficient magnitude, can cause respiratory failure.

The different etiopathogenic factors, already mentioned in the classification, will cause respiratory failure.

ratory, through certain pathophysiological processes that constitute, isolated or in association, something like common final pathways.

These main pathophysiological mechanisms are: hypoventilation, imbalance in the ventilation/perfusion relationship, intrapulmonary shunt or diffusion limitation, although the latter mechanism is less frequent, as a cause of significant hypoxemia.

Hypoventilation (ventilatory failure)

Hypoventilation implies that alveolar ventilation is not sufficient to cover the CO elimination needs, so paCO tends to rise. This hypercapnia causes respiratory acidosis and is associated with hypoxemia, because the decrease in alveolar oxygen pressure (PAO) caused by hypoventilation leads to a decrease in arterial oxygen.

That is, it basically causes hypercapnic-hypoxemic respiratory failure.

It occurs in multiple neurological disorders, respiratory muscle disease, other conditions of the thoracic bellows and chronic obstructive pulmonary diseases, which allows it to be classified into restrictive or obstructive type respiratory failure.

A. Restrictive.

In restrictive respiratory failure there is no obstruction of the airway.

Restrictive ventilatory insufficiencies can be due to nervous and muscular causes or thoracopulmonary causes.

Restrictive ventilatory insufficiencies due to nervous and muscular causes have hypoventilation as the only mechanism, but they are not accompanied by lung or thoracic injuries and that is why they can be easily corrected by assisted respiration.

They can be due to anesthesia, overdose of sedatives, neuromuscular diseases such as myasthenia gravis and Guillain-Barré syndrome, among other morbid conditions such as botulism, amyotrophic lateral sclerosis, severe spinal trauma and muscular dystrophies.

In the positive diagnosis, the clinical picture of the underlying disease, accompanied by cyanosis and decreased thoracic excursion, is of

Patients who suffer from restrictive respiratory failure due to thoracopulmonary causes are characterized by being polypneic and having variable degrees of physical disability.

What is striking is a notable decrease in the expansibility of the lungs due to disorders of the chest wall (kyphoscoliosis, obesity, Marfan, etc.), chest trauma, pleural disorders (effusions, pneumothorax, fibrosis) or lung disorders (fibrosis).

B. Obstructive.

Obstructive ventilatory insufficiency occurs due to increased resistance to gas flow in the airways (bronchial asthma, bronchitis, foreign bodies, tumors, among other causes).

Its fundamental manifestation is inspiratory dyspnea, when the obstruction is in the large airways (larynx, trachea) with cornage and indrawing; and expiratory bradypnea, if the obstruction is in small bronchi, accompanied by wheezing rales as in bronchial asthma, or with hoarse rales, if the obstruction is in larger bronchi.

In these cases, the work of breathing increases due to increased resistance to gas flow.

In these patients, of course, there is also hypercapnia (paCO, high) and hypoxemia (pa0, low) if a large part of the tissue is hypoventilated.

In addition to the characteristic bradypnea, spirometry reveals a decrease in maximum respiratory capacity and vital capacity and air trapping. The improvement obtained with bronchodilator drugs shows a spasmodic component. There is pulmonary hyperinflation and the residual functional capacity is increased.

In chronic cases, structural modifications of the lung (destruction of elastic fibers), airways and pulmonary vascularization occur that cause secondary pathophysiological alterations and add to hypoventilation.

Imbalance of the ventilation/perfusion ratio

Normally there is a balance in the distribution of alveolar ventilation and the corresponding blood perfusion. When this balance is altered, such that perfusion exceeds ventilation, it can cause hypoxemia.

Although in addition to the reduction in alveolar oxygen pressure (PAO,), the alveolar pressure of CO, (PACO,) increases, the final predominant result is hypoxemia. This is the case, given that increasing ventilation in regions where the balance between ventilation and perfusion is relatively normal leads to sufficient elimination of CO, and thus compensates for the elevation of PACO in the most affected areas.

This mechanism, which manifests as hypoxemic respiratory failure, is typically observed in moderate to severe bronchial asthma.

Intrapulmonary shunt from right to left

It can be considered as an extreme degree of ventilation/ perfusion imbalance, the clinical picture that

It occurs in alveolar areas that are still perfused and are no longer ventilated, due to collapse, flooding by mucus, cellular debris, pus or edema.
This results in the formation of a true intrapulmonary shunt from right to left. In this way, the venous blood that reaches the lungs from the right heart chambers returns to the left heart without having been adequately oxygenated.

This results in arterial hypoxemia that is resistant to treatment with high oxygen concentrations.

It is the main pathophysiological mechanism of hypoxemia in most severe acute respiratory failures, such as adult respiratory distress syndrome, severe pneumonia, and cardiogenic pulmonary edema.

Disturbance or limitation of diffusion

When there is involvement of the alveolar wall, the interstitium or both, as in the case of fibrosis, hypoxemia may occur. However, it is rare as a cause of clinically significant hypoxemia.

This happens because normally, the blood passing through the pulmonary capillary bed only requires a third of all available time, for almost total saturation of hemoglobin with oxygen. In this way, although there is limitation of oxygen diffusion, the total time available allows the hemoglobin to be saturated with Oxygen.

This disorder usually becomes evident during exercise, due to the rapid transit of red blood cells in the blood flow through capillaries depleted by interstitial involvement. It can also be evident at high altitudes, as a result of the lower alveolar oxygen pressure.

Mixed

In many cases there is more than one pathophysiological mechanism involved. This is the case of pulmonary fibrosis itself, in which the common mechanism of hypoxemia is given by ventilation/perfusion imbalance, associated to a greater or lesser degree with pulmonary shunt.
This association is also observed in pulmonary embolism.

On the other hand, the combination of hypoventilation and ventilation/perfusion imbalance is observed in chronic obstructive pulmonary disease (COPD) and very severe asthma.

Syndromography or positive diagnosis

Clinical picture

The patient may have a history of a previous chronic respiratory condition, a respiratory condition, acute illness, a systemic condition or other morbid conditions that can disrupt respiratory function, such as excessive ingestion of psychotropic drugs and polytrauma. Therefore, the clinical manifestations may be dependent, on the one hand, on the previous or concomitant underlying conditions, causative or predisposing, and on the other, on hypoxia and hypercapnia.

Although the diagnosis of respiratory failure is established by the study of blood gases in the laboratory, which allows hypoxemia and hypercapnia to be determined, there are symptoms and signs that depend on these alterations, and allow us to suspect them when they are detected in the clinical examination.

Chronic hypoxia results in neuropsychic manifestations, such as drowsiness, lack of attention, fatigue, delayed reaction time. The plethora, caused by erythrocytosis, is characteristic. However, chronic cardiovascular effects are generally minimal, although physical examination may detect pulmonary hypertension and even chronic cor pulmonale, with signs of right heart failure.

Acute hypoxia is basically expressed in relation and proportion to the abnormalities it causes in the function of the central nervous system and the cardiovascular system. Thus, according to the progressive worsening of hypoxia, symptoms and signs will appear in an approximate order, as follows: tachycardia, dyspnea in the form of tachypnea, anxiety, restlessness, impaired judgment, fear, headache, angina pectoris, confusion, cyanosis, arterial hypertension first and then arterial hypotension, bradycardia, seizures, myocardial depression and shock.

Chronic hypercapnia is manifested by headache, drowsiness, involuntary muscle movements, asterixis, papilledema.

In acute hypercapnia, depending on the excess of CO, and the rapidity of its accumulation, progressive alterations of the central nervous system (CNS) manifest: apprehension, drowsiness, lethargy, confusion, restlessness, tremor, headache, asterixis, papilloedema and coma. The cardiovascular effects vary depending on the predominance of vasoconstriction (due to generalized sympathetic activity) or vasodilation (due to local accumulation of CO2). There is usually tachycardia and sweating, but there may be hypertension, hypotension or normotension.

Acute hypercapnia causes depression of CNS activity by reducing the pH of the cerebrospinal fluid.
Therefore, mental disorders are more closely correlated with low pH and acidosis than with the C0 level. That is, we should speak of hypercapnia with acidosis.

Complementary exams

Determination of arterial blood gases. paO, low (less than 50 mm Hg) in acute respiratory failure (some place the limit at 55 and even 60 mm Hg) and paCO, high (greater than 50 mm Hg) in acute respiratory failure.

Chest x-ray. It may be normal or show diffuse interstitial infiltrate, diffuse alveolar infiltrate or localized alveolar infiltrate, depending on the underlying disease.

tiology

We can organize and summarize the causes, depending on whether it is chronic or acute respiratory failure, as well as whether the latter is hypoxemic or hypercapnic-hypoxemic.

Chronic respiratory failure

-Obstructive causes:

Chronic bronchitis.

Emphysema.

Cystic fibrosis.

Bronchiectasis.

bronchial asthma.

- Pulmonary vascular diseases:

Pulmonary vasculitis (collagen diseases).

Recurrent thromboembolisms (drug abuse, sickle cell disease and schistosomiasis).

- Diseases that cause infiltration of the parenchyma and/or interstitium:

Sarcoidosis.

Pneumoconiosis.

Idiopathic fibrosis.

Leukemia.

Hodgkin's disease.

Radiations.

Systemic lupus erythematosus.

Drug sensitivity.

Others.

Diseases that cause pulmonary edema:

Cardiogenic (congestive heart failure).

- Diseases of the pleura and chest wall:

Idiopathic or severe acquired kyphoscoliosis.

Massive pleural effusion or effusions with pachypleuritis (thickened, constrictive pleural layer).

-Abnormal respiratory center:

Primary hypoventilation syndrome or "Ondine's curse."

- Disorder of the spinal cord and peripheral nerves:

Poliomyelitis.

Polyneuritis.

-Muscle disorders:

Muscular dystrophy.

- Sleep apnea:

Massive obesity.

Chronic mountain disease.

Acute respiratory failure

-Obstructive causes:

Upper respiratory tract: swelling due to inflammation of the mucosa, due to infections, allergic reactions (laryngitis, tracheitis), and thermal or mechanical injuries; foreign body impact; tumors.

From the lower respiratory tract: swelling of the mucosa, secretions in the lumen or bronchospasm.

Examples: bronchial asthma, infections, bronchiolitis, or inhalation of chemical substances (nitrogen dioxide in "silo fillers disease).

- Pulmonary vascular diseases:

Pulmonary thromboembolism.

Fat embolism.

Embolism due to aggregates of platelets and fibrin, in disseminated intravascular coagulation (DIC).

Diseases that cause infiltration of the parenchyma and/or interstitium:

Pneumonias.

Infectious (the most common cause of parenchymal infiltration).

Inhalation or aspiration of toxic chemicals

Cas.

Immunological reactions to medications.

Parasite migrations.

Leukoagglutinins.

- Diseases that cause pulmonary edema:

Cardiogenic: acute myocardial infarction; acute left ventricular failure, due to hypertensive crises or arrhythmias; valvular heart disease.

Non-cardiogenic (due to increased permeability):

Adult respiratory distress syndrome.

- Diseases of the pleura and chest wall:

Chest wall injury: segmental fractures of several ribs or rib fractures, on both sides of the sternum.

Spontaneous or traumatic pneumothorax.

Brain disorders:

Impairment of CNS function, due to the use of sedatives or anesthetics.

Cerebrovascular diseases.

Tumors.

Infections.

- Disorders of the spinal cord and peripheral nerves:

Cervical or upper thoracic spinal cord injury.

Poliomyelitis.

Infectious polyneuritis (Guillain-Barré syndrome).

Toxic polyneuritis.

- Neuromuscular disorders:

Myasthenia gravis (in acute myasthenic crisis).

Acute hypoxemic respiratory failure -With previous lung disease:

Restrictive lung disease (previous pulmonary fibrosis).

Moderate to severe bronchial asthma.

- Without previous lung disease (although it can be added to a previous disease):

Adult respiratory distress syndrome.

Cardiogenic pulmonary edema.

Cardiogenic pulmonary thromboembolism.

Pneumonia/bronchopneumonia.

Acute hypercapnic-hypoxemic respiratory failure

-With previous lung disease:

Chronic obstructive bronchopulmonary disease (COPD).

Very severe bronchial asthma.

-With normal lungs:

Sedative overdose.

Neuromuscular disease (myasthenia gravis).

Guillain-Barré syndrome.

ADULT OR ADULT TYPE RESPIRATORY DISTRESS SYNDROME

Since Ashbaugh and collaborators described adult respiratory distress syndrome (ARDS) in 1967, it has taken center stage in modern clinical and medical pathology, constituting an im-

leading cause of morbidity and mortality in intensive care units.

With various and even colorful synonyms, this syndrome can be defined, according to its first description, as an especially severe acute respiratory failure, characterized by intense dyspnea, tachypnea and hypoxemia refractory to treatment with high concentrations of oxygen, with decreased lung compliance and the presence of diffuse alveolar infiltrates on radiology, and which can be due to multiple etiologies.

Syndromogenesis or physiopathology

The various pathogenic factors of the syndrome, which can be direct, as in lung contusions and aspirations, or indirect, as in sepsis, poisoning and polytrauma, cause a disturbance in lung function. This occurs from endothelial dysfunction caused by a series of inflammatory mediators, such as O radicals, cytokines, adhesion molecules or thromboxanes, which produce non-cardiogenic edema.

Pathologically, there is an increase in weight and consistency of the lungs, as well as a diffuse alveolar lesion with alveolar edema, interstitial inflammation, hyaline membranes and interstitial fibrosis, which are non-homogeneously distributed. To these alterations are added those of secondary sepsis and those derived from mechanical ventilation.

All of this results in a decrease in lung compliance or distensibility, pulmonary hypertension, and an alteration in the extreme ventilation/perfusion relationship, which gives rise to an intrapulmonary shunt and severe hypoxemia.

Syndromography or positive diagnosis

Clinical picture

History of acute illness or injury that directly or indirectly damages the lungs, especially sepsis, trauma, prolonged or profound shock, fat embolisms, massive transfusions, among others. 12-24 or 72 hours after the initial injury or illness, or 5-10 days after the beginning of an infection, the manifestations of acute respiratory failure appear, given by: dyspnea; tachypnea (respiratory rate greater than 30/min);

hyperventilation; noisy expiration; intercostal and suprasternal indrawing; cyanosis; conjunctival and axillary petechiae, in fat embolism; rales may or may not be auscultated; CNS alterations; there may be clinical signs of shock.

Complementary exams

Chest x-ray. Diffuse bilateral alveolar infiltration (pulmonary edema without cardiomegaly).

Analysis of arterial gases. paO, low, with increasing decrease, despite high concentrations of inspired oxygen; paCO, normal or low.

tiology

- Infectious (according to some authors the main medical risk factor is sepsis):

Sepsis syndrome (infection with systemic complications, such as hypotension, metabolic acidosis, or both).

Pneumonia/bronchopneumonia.

- Severe pulmonary and extrapulmonary trauma.
- Aspiration of liquids: gastric juice, fresh and salt water (incomplete drowning syndrome).

Body burns.

- Drug overdose: heroin and other opiates, salicylates, barbiturates, propoxyphene.

-Inhaled toxins: high doses of oxygen, smoke.

- Toxic ingestion: paraquat.

- Metabolic disorders: pancreatitis, uremia.

- Others: intracranial hypertension, eclampsia. Postcardioversion.

CIRCULATORY SYSTEM. HISTORY AND MAIN SYMPTOMS

37

ANAMN SYSICAL DATA IN CIRCULATORY SEMIOLOGY

In the study of the semiology of the circulatory system, the collection of anamnestic data is of great importance. For its study we will stick to the medical record models that we use in our environment, both in primary care and secondary care.

AGE

Age is a factor of some importance. Of course, in circulatory conditions there are processes that occur in early ages, and others that are seen in advanced ages. In the early ages of life, it is the time when conditions of congenital origin are most frequently recognized, although not exclusively, because congenital lesions can also be identified, both in puberty and in adulthood. Of course, most cases are discovered at an early age, such as congenital pulmonary stricture, persistent Botal foramen or Roger's disease (ventricular septal defect), etc.

On the contrary, at advanced ages or above middle age, other conditions appear. They are those disorders that are characterized by degenerative disturbances of the circulatory system and correspond to the process of involution (regressive evolution): arteriosclerosis and atheroma, which affect the arterial vessels or the heart itself.

In the middle age of life, of full activity, intense struggle, great efforts, above all, intellectual efforts and exposure to great emotions, it is also a time when certain cardiac disorders frequently occur, particularly cardiac infarction.

SEX

Sex is not a predominant factor, but it can be seen that cardiac infarction and chronic cor pulmonale (pulmonary heart) are much more common in men.

It is also important in terms of events that are specific to the female sex: such as pregnancy and childbirth. Both the first and the second are capable of recognizing a valve lesion, which until then had remained hidden. Here, pregnancy, and about

All the effort of childbirth causes the symptoms of heart failure to begin. That injury had remained compensated or the heart had adapted to it without presenting symptoms.

During pregnancy or childbirth, this adaptation is broken and an accident occurs, sometimes of a paroxysmal type; These are the so-called gravid-cardiac accidents. On other occasions, heart failure begins in any of its progressive forms.

0CUPATION

Certain occupations can cause chronic lung lesions (silicosis, asbestosis, siderosis) and thus be the cause of the development of chronic cor pulmonale.

Sinus bradycardia of 40 beats per minute is common in athletes, as enlargement occurs through a compensation mechanism. This fact is important to know so as not to make a false semiological interpretation.

HISTORY OF THE DISEASE

Next anamnesis

We will not tire of insisting on the importance of a good interview with a patient and that is why we will stop by giving some typical examples. On many occasions, the story of the suffering that makes up a person's illness constitutes almost the entire diagnosis, even though the physical examination does not provide any information.

On other occasions, the most frequent, the disorders that the patient suffers from also have their expression in the physical examination. Therefore, these data are of the greatest importance. First of all, we will be interested in knowing how the current illness began, an aspect that can take two main types. It should be understood well that when we say modes of onset we are referring to mode of onset in clinical settings, that is, ways in which the symptoms of the disease manifest. Thus, circulatory conditions adopt two main forms of onset: abruptly, in which suddenly, a subject in an apparent state of health presents manifestations of a circulatory process, for example; or, on the contrary, in a gradual progressive way in which the onset of the symptoms becomes slower and first symptoms of less importance appear and then little by little these symptoms become more intense or new ones are added to them, in a more or less ascending curve. Next, let's look at some examples that can establish these ideas.

Abrupt onset cases

Let's take angina pectoris as an example.

Generally it is a healthy subject, almost always thick, who suddenly presents with great intensity pain that, sometimes, can end his own life.

And see also how this case in which a painful manifestation (subjective symptom) reflects a pathological process, may nevertheless, during the examination, not offer signs that reveal the injury.

Another example is that of a subject who, being in good health and with the same general conditions that we have described in the previous one, wakes up one night with an intense feeling of lack of air, almost always accompanied by cough, which at first has little expectoration and which later produces aerated expectoration; This is a crisis of acute lung edema. The physical examination confirms the process and action is taken with the necessary urgency.

A third example is that of a subject who, feeling perfectly well, will momentarily feel a precordial beat, and following this sharp thud, he notices that his heart rate is altered, his heart beats at high speed and this sensation lasts for a variable time, which can be minutes, hours or days, and, for the moment, another sudden sensation like a stop in the precordial region ends the subjective sensation: this is the case of tachycardia. paroxysmal.

Progressive gradual onset case

Other times the beginning of these processes is slower. This is the case of an individual who has been feeling bad. If you are an ordinary type of individual, you notice that, for the first time, one day you cannot reach the bus on time, and you run after it and suffocate. He had dyspnea, which is physiological, but when it is exaggerated it is not. The dyspnea this time was very intense and annoying and the individual took longer to recover than usual. Other times it will no longer be that effort, but when climbing the stairs at home or at work, you notice that you arrive at the top very suffocated and with intense dyspnea. Days later he can no longer climb the entire staircase at once and is forced to stop at a landing. And so on, the symptoms gradually increase, until there comes a time when he goes to the consultation and reports that respiratory discomfort accompanies him almost constantly. The act of getting up, dressing, bathing, any very small effort causes dyspnea, until, finally, it becomes permanent. Here you can see how, gradually, from this first, latent phase, we have moved on to this other phase in which the dyspnea is permanent; how, also little by little, dyspnea has appeared on occasion of lesser efforts until it becomes permanent, accompanied by palpitations, etc. This is the case in which the onset of the condition is slow and progressive and not paroxysmal as in the previous cases.

We have detailed these examples a little to clarify the diagnostic importance they have and so that the value of the interrogation that we provide can be appreciated.

places you in a position to achieve valuable diagnostic guidance. If you know how to collect data, if you know how to extract it from that large volume of things that the patient tells us, you have taken the first step on the path to diagnosis.

The other methods must confirm the suspicion that the interrogation may have aroused.

The evolution of the current disease is the next aspect that we are going to consider. The patient has already told us how his suffering began, now we are going to be interested in how it has evolved. Using the same examples we can see different forms of evolution. There are circulatory processes that appear suddenly, last a short time and disappear for a long time, in which the patient feels well, only to appear again; For example, the paroxysmal tachycardia already mentioned. You notice this character of abrupt beginning and abrupt ending, and then a variable time interval of well-being. There are times when they go years without having another crisis. This data is going to be very valuable.

The evolution is also interesting, for example, in angina pectoris and heart attack. Angina pectoris is a sudden pain that usually disappears quickly, when it does not end the life of the subject.

On the other hand, in cardiac infarction the evolution is very characteristic. There is precordial pain, with similar characteristics, but its duration is longer, and this process lasts for hours and days; It is a type of intense, constant pain, and then if the subject does not die, we observe how the pain decreases and the crisis disappears, but not before determining fever and other characteristic symptoms. As we see, they are two different forms of evolution, which are very useful to distinguish one case from another.

On other occasions the evolution of the disease will be through successive outbreaks. This is the case of rheumatic endocarditis where a febrile process begins, and quickly, after a few days, it begins to give subjective circulatory manifestations. Afterwards the subject remains relatively well and after a variable time the infectious manifestations, fever, etc., will return, and thus a series of successive outbreaks, which of course can leave consequences.

On the other hand, if it is the history of infectious endocarditis of the ulcerovegetating type caused by a streptococcal infection, then its evolution continues without pause. The process progresses and can lead to death if adequate therapy is not instituted.

See, then, how the interrogation, both in the form of the beginning of the process and in the form of its evolution, acquires an extraordinary value in diagnostic orientation.

Remote anamnesis

As we know, it provides data on the subject's personal history and family history.

What value do these two sets of data have? Next, we will see it.

Personal pathological history

First you have to go look for (because the patient is not going to relate them, unless he is an educated man) the etiological factors. One of the most frequent etiologies of cardiac lesions is rheumatism, rheumatic infection.

Acute articular rheumatism is not always recorded in the clinical history in a typical way. Of course, if the affected subject or family members report that this patient has suffered from a febrile infectious process, which had localizations in his joints, which began with some pharyngeal manifestations, we will have a classic type that does not offer difficulty. But the story is not always so clear; On the contrary, in our environment this type of rheumatic history, far from being the rule, is the exception. We might think that rheumatism almost does not exist in our country if we are guided by the previous reference. On the other hand, if we investigate, we will know that the patient, at one point in his life, had chorea (nervous manifestations characterized by movement disorders); or we will simply know, and this is more common, that the subject has suffered from throat attacks, febrile angina with pain when swallowing, inflammation of the local lymph nodes and fever. And this febrile angina is what reveals the rheumatic infection. Perhaps the patient remembers that, indeed, during those febrile angina, he had bone or joint pain. Other times he will tell us that he never had localized pain, but rather it was muscle pain. This will be enough to identify the rheumatic condition.

Perhaps one of those febrile processes, seemingly banal, was a rheumatic infection. Other times in the presence of young people, rheumatism can be hidden behind the so-called growing pains.

With increasing longevity, arteriosclerosis is the most common etiology of heart disease, followed by arterial hypertension. In the past, rheumatism and syphilis were considered the most frequent etiological factors. The latter gives very important manifestations in the circulatory system, especially in the aorta. Let us point out its special form, which affects the middle layer and is preferably located above the aortic sigmoid, giving rise to suprasigmoid aortitis, with dilation of the vessel due to damage to the elastic layer and can lead to something else: the aneurysm, which of one type or another, are nothing more than dilations of the vessels, circumscribed in the shape of a spindle. These aortitis usually occlude the entrance to the coronary arteries. syphilis

It can extend to the coronary arteries, affect the myocardium itself and form gonmas that are located at the level of the bundle of His, hence the etiological importance of syphilis, the existence of which we will know from the patient's history.

We will learn that this subject had a chancre at some point in his life, and, if he remembers, he will say that he had regional lymph node infarctions and, above all, he will inform us that he had spots on his body. If he was better cared for and at that time the ultramicroscopic investigation was carried out and a pale spirochete was found, we are completely sure that he had syphilis. Likewise if it is known that he presented a positive serology in the blood. There are times when none of this is recorded, the man denies that he had a chancre, because sometimes the penetration of the infecting agent is done through insignificant erosions. As we see, there are many indirect ways to identify the process.

Other pathological antecedents that we collect are important in the production of circulatory conditions.
We are going to record high blood pressure and arteriosclerosis in the patient's history. These are subjects who will tell a story of headaches, cramps, loss of memory or loss of mobility in the extremities, some with manifestations of excess urine elimination during the night, which will generally be observed in obese subjects, who abuse not only the table, but also alcohol, and this hypertension will be a determinant of early involution of the arteries, of arteriosclerosis.

On other occasions they are disorders that affect the pulmonary circulation, which cause the pressure in the pulmonary artery to increase, which we call pulmonary hypertension or small circulation hypertension. Chronic lung conditions, pulmonary emphysema, fibrosis or sclerosis of the lung, can cause it.

The blood pressure in the pulmonary artery rises and as it rises, the right ventricle has to work harder and, therefore, dilates; This dilation becomes frankly pathological and compromises its function.

Lesions of the mitral valve, such as mitral stricture, which do not allow good emptying of the left atrium or the pulmonary veins, create venous hypertension in that territory, which in turn determines greater resistance in capillary circulation and, therefore, increased pressure in the pulmonary artery.

And finally, these hypertensive alterations of the pulmonary circulation can be a consequence of other disorders of the architecture of the thorax, such as kyphoscoliosis.

Gender of life

The individual who leads a disordered life, who abuses his strength and exaggerated taste for the pleasures of life table, alcohol, tobacco and others, is more exposed to the involutional alterations of sclerosis. And this will explain the appearance, at a relatively early stage of his life, of manifestations of one type or another. If we add to this the way in which life unfolds, in which the effort is intense, especially intellectual, the emotional tension is great, because the struggle itself determines it, in any activity, it is understood that certain processes such as heart attack are characteristic of that stage.

Feeding

We already pointed out its importance. Those who eat excessively, especially by abusing carbohydrates and fats, are predisposed to these circulatory disorders.

Toxic habits

Among these habits we have coffee and tobacco.
They play an important role by acting in the production of aortic processes and precordialgia. There are subjects who, when they abuse the tobacco habit, suffer from chest pains and these disappear when they abstain from smoking. There is also a higher incidence of heart attack among smokers.

Profession

Especially those exposed to emotional tensions, among which is the medical profession, because although the common people believe that the doctor is hard-hearted and has an imperturbable serenity, he does not know the emotions and anguish he experiences, because he has a great sense of responsibility; This influences the appearance of coronary disorders.

Other times this profession factor is of the type of professional poisoning. Lead poisoning has among its manifestations high blood pressure. These poisonings occur in subjects who work and do not adopt established occupational hygiene measures.

Hereditary or family history

Although there is no inheritance of heart diseases, there is a predisposition that, when transmitted from parents to children, favors the appearance of certain diseases. Let us cite as examples, high blood pressure, arteriosclerosis and rheumatic diseases. Ayman has verified that when one of the parents has essential hypertension, this disease appears in 20% of the offspring, a figure that rises to 40% when both parents suffer from the disease.

When the mother has suffered from certain viral diseases during pregnancy (rubella, measles, etc.), alterations in the development of the heart may occur and the child is born with congenital heart disease.

MAIN SYMPTOMS OF CIRCULATORY CONDITIONS

The main symptoms are:

- Pain.
- Dyspnea.
- Palpitations.
- Brain circulatory manifestations.

PAIN

Generalities

Cardiovascular pain is defined as any pain caused by conditions of the heart and blood vessels; However, in general, these pains refer to those that originate in the heart or in the great vessels that emerge from it, and that are located almost fundamentally in the precordial and sternal regions.

It is worth noting here that from a strictly scientific point of view, cardiovascular pain can be:

1. Central type, that is, it originates or is produced at the level of the heart or the great vessels, and is almost always located in the precordial region or its surroundings.
2. Peripheral or peripheral vascular type, which occurs in conditions that are located at the level of the arteries and veins of the limbs, producing a type of pain at this level that we will study later.

We must also point out and clarify that pain in the peripheral vessels can also occur at the level of the different organs when any of the arteries or veins of these organs (spleen, liver, intestine, etc.) suffer a sudden partial or total occlusion mechanism. These pains, however, will not be studied in this chapter since they almost always correspond to the pathology of the mentioned organs, and are specifically studied at the level of the viscera of the digestive, hematopoietic, etc. systems.

We are going to refer only to central cardiovascular pain, which is the most important from the point of view of the cardiovascular system. Peripheral pain will be treated in the peripheral vascular system."

Before entering into its consideration, it is necessary that we remember the innervation of the heart. To do this, we are going to analyze the pathways of cardiac sensitivity and study the pathophysiology of cardiac pain.

The heart is innervated by the sympathetic and parasympathetic systems. The nerves of the heart are part of the vagus nerve and the sympathetic system.

There are sensitive nerve endings, that is.
afferents, in the myocardium, pericardium, endocardium, coronary arteries and in the aorta, extraordinarily increased in the latter; These fibers come together and form the coronary and cardiac plexuses, which can be superficial and deep, that is, the afferent fibers coming from the endocardium, the myocardium and the pericardium, and in greater quantities from the coronary arteries and the aorta at the beginning, form, upon leaving that site, the superficial and deep coronary and cardiac plexuses. Three nerves originate from here, the so-called superior, middle and inferior cardiac nerves.

The superior cardiac nerve ends in the superior cervical ganglion, and does not contain sensory fibers; while the middle and lower cardiac nerves, which do contain sensory fibers, go to the middle and lower cervical ganglia or stellate ganglion: but as these cervical ganglia lack rami communicans to connect with the spinal cord, these fibers descend through the sympathetic chain until they reach the first dorsal ganglia (first to the fourth or fifth), where connections can be established with the spinal cord through the rami communicans.

It has also been proven that there are thoracic cardiac nerves that cross the posterior mediastinum to go from the heart to the superior thoracic ganglia directly; That is why mediastinal conditions can cause pain at the precordium level through a direct mechanism.

The sympathetic nerves, mainly the postganglionic fibers, arise from the three cervical ganglia and the five upper thoracic ganglia; These nerves are: the upper cervical cardiac, the middle cervical cardiac and the lower cervical cardiac.

The thoracic cardiac nerves arise from the thoracic ganglia of the sympathetic chain.

The cardiac branches of the vagus nerve begin: in its cervical portion, the superior cardiac branches; in its thoracic portion, the middle cardiac branches and in the recurrent laryngeal nerve, branch of the vagus, the lower cardiac branches.

For pain to exist there must be an adequate stimulus such as ischemia, which produces, according to some, the release of certain substances that stimulate the nerve endings.

Inflammations of the serous membranes or distentions of a hollow viscus can also cause pain.

The usual stimuli, both physical and chemical, do not produce pain.

In 1970, Vane presented the theory about the action of certain non-steroidal anti-inflammatory substances such as aspirin and indomethacin, which inhibit the synthesis of prostaglandins, which seems to offer an explanation

satisfactory explanation of the mechanism of these therapeutic agents widely used in the relief of pain caused by inflammation. Thanks to this discovery, it is considered that cardiovascular pain can be caused by the formation of small amounts of prostaglandins and their precursors, which act by sensitizing pain receptors.

Currently, a double mechanism is accepted to produce visceral pain not only in the heart, but in a general sense: one is referred pain, according to Mackenzie Hess and Ross, which gives rise to the viscerosensory reflex, and another is the direct mechanism, in which the sympathetic fibers would also include current sensory fibers, such as cutaneous fibers; This would be direct visceral or pure visceral pain. In relation to the first, that is, referred pain, it is currently accepted that the sensitive nerves of the skin and the sensitive visceral nerves carry their respective impulses to the common sensory neurons and are added there. In a normal state, this sum of impulses does not reach the excitability threshold of the secondary neurons of the thalamus and cortex, which is why pain is not produced; But in a pathological state, the greatly increased visceral stimuli, added to the cutaneous ones, can exceed the threshold and therefore produce referred pain.

Naturally, this also depends on the state of previous excitability of the neurons; Sometimes it lowers the threshold, as happens in the pain mechanism of nervous and apprehensive subjects, which sometimes makes them suffer in almost normal conditions.

Central cardiovascular pain

We will now consider the most frequent central cardiovascular pains; They can adopt three modalities:

- Anginal pain.
  Simple precordial pain.
- Precordial pain.

Anginal Pain

Concept

It can be defined clinically as precordial pain frequently radiating to the left arm, neck and jaw on the same side:; of a constrictive nature, triggered by effort and frequently accompanied by anxiety or a feeling of imminent death.

semiography

Location. More common in the precordial region. Patients indicate this by placing their open hand on the precordium, not with the tip of their index finger.

Intensity. It is large and deep in nature, although pain of minimal intensity has been reported, also called angina minor by the classics.

Quality. It is almost always accompanied by a feeling of anguish or imminent death with retrosternal oppression. The patient reports as if a bite had stopped in his esophagus, or as if his throat had become knotted; This constriction can also appear in the places corresponding to the irradiation, such as: the neck and limbs, in these the patient reports the sensation of strangulation, as if he were wearing a bracelet. Sometimes you just have various paresthetic sensations.

Irradiation. It is commonly carried out towards the shoulder, arm, neck, lower jaw, ear canal, cheeks, back, that is, to the back or also in a cross bar.

The main radiation, however, extends through the left arm, particularly the ulnar portion, and reaches the little finger and ring finger of the hand (Fig. 37.1).

Its duration depends on the type of injury that has given rise to the pain, and may be temporary and only last a few seconds (angina pectoris), or, on the contrary, more permanent or sustained for hours or days (myocardial infarction).

Appearance conditions. In general it appears almost always linked to the effort on the part of the heart; This is seen more frequently in physical exercise, or when there is an increase in the functions of the digestive organs - for example, during digestion -, or sometimes during intercourse, or sometimes when forced urination occurs due to dysuria, or during defecation in constipated subjects.

It can also occur due to the effect of emotions of any kind. Likewise, cold has also been invoked as a factor that produces anginal pain.

Associated symptoms. It is frequently accompanied by symptoms mainly from the digestive system, such as gastric fullness or distension. Sometimes nausea or vomiting that can confuse the diagnosis. It is also accompanied by paleness and high blood pressure in angina pectoris. And, on the contrary, it frequently presents hypotension, coldness and sweating (shock) in cases of heart attack.

The electrocardiographic examination in most cases does not reveal morphological alterations when it comes to a case of angina pectoris and does in cases of coronary thrombosis.

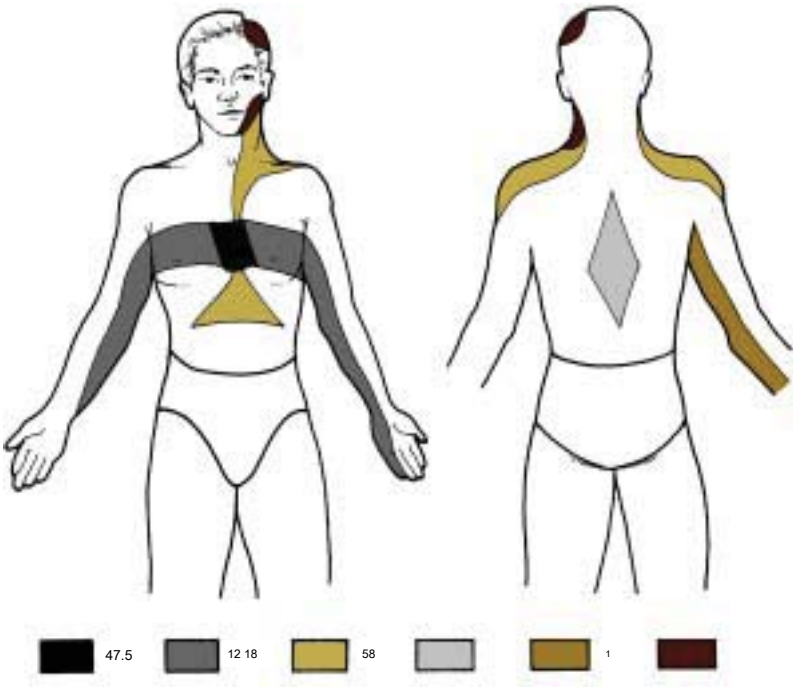


Fig. 37.1 Radiations of anginal pain.

Semiogenesis or pathophysiology

Experimental cauterization and distention of the heart has been shown to be painless, although pain is the dominant symptom of angina and heart attack.

The fundamental cause of this pain, as has been demonstrated, is due to ischemia of the cardiac muscle.

It is convenient at this moment to do a quick reminder of the coronary circulation, to perfectly understand the modalities that can produce, in the clinic, anginal pain or heart attacks, which are due to different pathological entities.

Normal coronary flow is around 200 mL per minute, of which approximately 70% is equivalent to diastole and 30% to systole. This coronary flow is the result of the tension gradient that exists between the blood of the aorta, where the orifice of origin of the coronary arteries is located, and that of the coronary venous sinus and the Thebesian veins, whose pressure depends on the blood found in the right atrium where they empty.

It will thus be understood that aortic insufficiency, by decreasing intra-ortic pressure, reduces coronary output. Aortic stenosis also reduces mean intra-ortic pressure and coronary flow, easily causing angina.

In mitral stenosis with great pulmonary hypertension, the pressure in the right cavities and in the coronary sinus increases simultaneously, and at the same time the cardiac output decreases, both due to the mitral barrier and due to the reduction of the pulmonary arteriolar bed, which produces a low intraortic pressure; The same occurs in severe heart failure. In myocardial hypertrophy there is a relative ischemia due to the exaggerated development of muscle fibers eager for oxygen and other nutritional elements that cannot be supplied by the coronary circulation, since this does not increase in parallel, therefore there is a relative ischemia.

Rhythm disturbances, such as paroxysmal tachycardia, generate ischemia by virtue of the fact that the marked cardiac acceleration depresses, on the one hand, blood pressure and, on the other, decreases the duration of diastolic rest in the unit of time; We already said that the coronary network fills more during diastole. Furthermore, the increase in the number of contractions leads to an increase in the nutritional demands of the heart.

Mechanical occlusion of a coronary artery is also a cause of anginal pain. This occlusion can be caused by thrombosis or more rarely by embolism.

In the case of thrombosis, the arterial vessel has its endothelium inflamed, as occurs in arteritis or in an arterial degenerative disorder (sclerosis with atheromatous plaques). In addition to narrowing the lumen of the vessel, which is the cause of ischemia, blood coagulation may occur at the site of one of these injuries, obstructing the lumen of the vessel, and the territory that it irrigates may be totally and definitively deprived of its blood supply. This is the mechanism of thrombosis, in this case, coronary. But it may be that the vessel, whether or not affected by some process, reaches an embolus through the bloodstream that comes off from any place, and this embolus becomes lodged in the vessel, obstructing it, resulting in

resulting in definitive ischemia. Therefore, both cases can be found: due to coronary thrombosis, or due to coronary embolism.

In one or another mechanism, the certain fact is that this vessel, whatever it may be, is totally and definitively occluded and the area of the myocardium irrigated is ischemic and gives rise to pain. There is absolute ischemia of a permanent nature, and this area is called the infarction zone. That is heart attack, and this type of prolonged pain corresponds to this process.

This infarct zone suffers all the alterations consequent to permanent ischemia and evolves towards a process of necrobiosis that culminates with the local formation of a plaque of fibrous tissue (if it does not first lead to heart rupture); This plaque can give way over time and cause a heart aneurysm.

As can be seen, there is not a single case of angina in which absolute or relative ischemia of the cardiac muscle does not play a role.

Semidiagnosis

We must consider three fundamental types of anginal pain:

- Acute coronary insufficiency without occlusion.
- Pain from angina pectoris or angor pectoris.
- Myocardial infarction pain.

Acute coronary insufficiency without occlusion. It is produced by a sudden and accentuated coronary irrigation deficit, without vessel occlusion.

Patients with chronic coronary insufficiency may also encounter various circumstances that trigger acute coronary insufficiency, for example: great exertion, profuse bleeding, traumatic or surgical shock, or large tachycardias. Thus, acute coronary insufficiency without thrombosis is established when there is a sharp and sudden drop in the pressure gradient due to a drop in blood pressure. It is also more common in cases in which chronic coronary insufficiency is associated with this fact.

Of course, if the triggering factor of acute coronary insufficiency without occlusion persists in its effects, necrotic lesions are generated, but different from those produced by true occlusion. Necrotic lesions consist of small, disseminated, numerous, non-confluent or transmural areas, preferably located in the subendocardium or papillary muscles.

Pain from angina pectoris or angor pectoris. There is a tendency to reserve the name angina pectoris itself for this type; it is also called angina.

of effort because it is almost always the effort that precedes the pain.

It lasts a very short time, several seconds or at most a few minutes.

The pain is fleeting, it almost always occurs due to effort, an emotional state, but this is not an exclusive nature, because there are times when this type of fleeting pain also occurs during rest.

The ischemia that occurs is transient.

We already reviewed the semiogenesis of the pain of angina pectoris or angor pectoris in the semiogenesis of anginal pain in general.

Myocardial infarction pain. It is due to an embolism or thrombosis that produces an occlusion of the coronary arteries.

The duration is longer, usually much more than minutes, lasting hours, and ischemia causes permanent injuries.

It can appear at any time, at rest or when getting up.

The pain is intense and prolonged, which is its fundamental characteristic, and then a series of manifestations are seen that will inform us of the existence of a myocardial infarction: shock, nausea, vomiting, moderate fever. The semiogenesis of myocardial infarction pain was previously described.

The differential diagnosis between the different types of anginal pain is made by the duration of the pain, the conditions of appearance and the clinical picture that accompanies the pain. It is important to remember that anginal pain due to infarction can be accompanied by shock, nausea, vomiting, moderate fever, pericardial friction rubs, especially in anterior infarction, and rhythm disturbances. In addition, there is leukocytosis with polynucleosis, increased crytrosedimentation and elevated levels of glutamic-oxaloacetic transaminase.

However, in acute coronary insufficiency without thrombosis or occlusion, the shock phenomenon may have been the triggering cause and not the result of it.

The electrocardiogram helps extraordinarily and, in general, T wave alterations are observed spontaneously or by the Master's test of effort or exercise, in cases of angina pectoris; alterations not only of T, but also of the ST segment indicating the lesion and sometimes areas of necrosis characterized by Q or QS waves in acute coronary insufficiency without occlusion; and all the alterations described in much greater intensity and progressively evolving in myocardial infarction.

Simple chest pain

semiography

Simple pain is generally characterized by superficial pain, of moderate intensity and little spread.

Semidiagnosis

This type of pain occurs in conditions of the pericardium, in aortic conditions and in dilation of the cardiac chambers (Fig. 37. 2).

Conditions of the pericardium. It is observed in cases of acute pericarditis (almost always rheumatic), in which it is located in the middle or lower portion of the sternum, lasting as long as the process continues.

These pains have been subdivided by the Institute of Cardiology of Mexico into: pleuropericardial pain and mediastinal pericardial pain, considering that their semiogenesis involves the spread of pericardial inflammation, sometimes to the pleura, and on other occasions to the mediastinum. At the present time this is the most accepted conception.

Aortic conditions. Whether it is syphilitic aortitis or an aortic aneurysm, the pain is almost always located in the upper portion, near the sternal manubrium, or in some of the projections of the arch. At present, it is considered that aortic pain can be of two types:
aortomediastinal and aortoparietal.

It is called aortomediastinal pain, when it only invades the nerve fibers of the neighboring structures of the mediastinum and can improve precisely with antisyphilitic treatment, as has been seen repeatedly, when this is the cause of painful aortitis. It would therefore be aortomediastinitis, which yields to treatment due to regression of the mediastinal component.

In cases of aortoparietal pain, where there is invasion of the chest wall, sternum, ribs, vertebrae, nerves and muscles, it is a much more constant pain and is exacerbated with different movements.

Finally, it would be necessary to consider a special variety of aortic pain and that is what occurs in the so-called dissecting aneurysm of the aorta. In these cases, very intense pain can be found at the level of the sternum, or in the posterior region of the thorax, which radiates to the entire thorax, both arms and even the lower limbs; It is of great intensity and subsides when the rupture of the aneurysm causes the expulsion of the blood contained in the walls of the aorta towards the lumen or outwards. This disease is due to necrosis or cystic degeneration of the tunica media of the aorta, which causes the detachment of its wall, from the birth of the vessel to its bifurcation on some occasions.

Dilation of the cardiac chambers. A third group of pains is pain due to dilation of the cardiac chambers, particularly the left atrium and right ventricle.

Pain in the left atrium, which has been called atrial side tip by Vaquez, is a pain located in the posterior region of the thorax, to the left of the spinal column, that is, in the left scapulovertebral region.

The Mexican authors consider:

1. That dilations of the left atrium are found in some diseases that are not accompanied by pain.

2. That more or less intense pain occurs on other occasions when the increase in the left atrium is not very marked.

This has made them think that the pain caused at the level of the lateral tip of the ear is rather due to irritation of the mediastinal fillets and the nerves that go directly from the spinal cord to the heart through the posterior mediastinum.

The other type of dilation of the cardiac chambers, which in this case corresponds to the right ventricle, is the so-called painful epigastric beat of Brum, which is located in the highest portion of the epigastrium, below the xiphoid appendix.

These dilations and pain occur in cases of hypertrophy and dilation of the left atrium due to lesions that affect the mitral valve, such as stenosis or mitral insufficiency. The dilation is much more marked in mitral stenosis.

Pain due to dilation of the right ventricle will be present in pulmonary hypertension, due to any mechanism established in it.

Finally, we will point out another type of simple pain, which is that which is produced in certain subjects (especially sensitive and nervous) by extrasystoles that generate a fleeting pain as if it were a sting with a knife or a pin.

Precordial pain

Let us now consider the third group of chest pains; They are called precordial pain or cardiothoracic pain.

semiography

They can be located at any point in the precordial region and sometimes outside it; most frequently to the left of the sternum, around the tip; rarely seen

A LAN R SMA D S CANT A RT CO can cause sudden, tearing retrosternal pain that can mimic the pain caused by myocardial infarction.

B LA SCL ROS S CORONAR A causes paroxysmal pain, generally following physical exertion, which may radiate to the shoulders, arms or neck

C LAH RNAD AFRAGM T CA usually causes pain in the lower retrosternal region

The pain becomes more intense in a horizontal position and with respiratory movements

D LAP R CARD T S associated with involvement of the adjacent pleura causes pain that becomes more intense with cardiac displacement

LMOCARD 0 causes intense and prolonged pain, which does not decrease in intensity with rest; generally there are also manifestations of circulatory collapse

F LA ARTTRSD ART C LAC ON S CHONDROCOSTAL S and concomitant muscle spasm may cause diffuse pain; this occurs more commonly in middle-aged or elderly people.

G CABBAGE CST TS can cause pain radiating anteriorly to the left shoulder

Frequently the conditions of the gallbladder and arteries occur simultaneously

H LA PANCR AT TSAG DA can cause epigastric pain that is accompanied by manifestations of circulatory collapse, thus simulating a myocardial infarction.

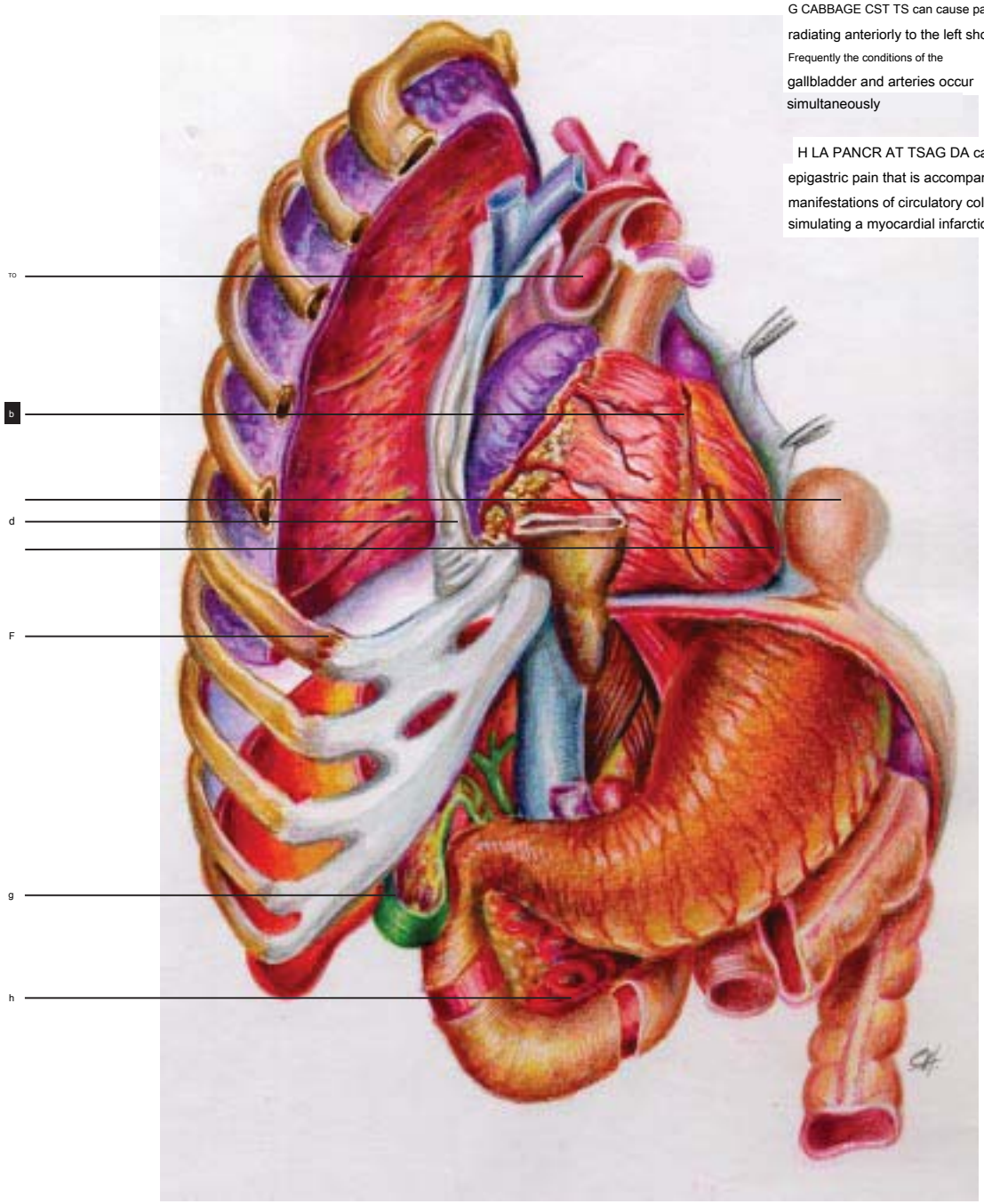


Fig. 37.2 Causes of chest pain.

ces on the lateral aspect of either hemithorax.
Although they are usually localized, they can cover a larger area and even radiate to the left shoulder or the left upper limb and the neck, as happens in angor. They are frequently accompanied by cutaneous hyperesthesia, which is bothersome even when the shirt rubs, in most cases they manifest as sharp pains (pricks) or fleeting pinches, which last a few seconds and occur isolated or are repeated several times; However, on other occasions there is a dull pain, without any special modality, or a burning pain or a painful discomfort that is difficult to define, and in such cases they may be of longer duration and continuous, and be accompanied by hyperesthesia.

Precordial pain is not related in its appearance to effort, although it may occasionally occur during exercise, but it never strictly obeys the law of effort and rest as occurs in angina pectoris; On the contrary, it is common for them to be more frequent and tenacious at the end of the daily work, when the patient's mind is less busy and the patient can better concentrate his attention on the symptoms that afflict him.

These pains are very frequently accompanied by nervous dyspnea, palpitations and other cardiac and extracardiac symptoms, which reveal a neurovegetative imbalance.

We have grouped here different types of pain that can occur in the precordial region and which have been precisely what we are considering under the name of precordial pain.

Semidiagnosis

In this sense, the following varieties should be considered: psychogenic pain, regional cellulitis, osteoarticular lesions, intercostal neuralgia, gallstones, gastric dilation or aerogastria, spasms of the cardia, etc.

DYSPNOEA

This symptom has been studied in the respiratory system and, therefore, we will limit ourselves here to specifying the data of semiological interest of the dyspnea presented by patients with the circulatory system, that is, cardiac dyspnea.

We will begin by studying in a general way the semiogenesis of dyspnea in cardiac patients and then its different modalities.

Semiognesis or physiopathology

In most cases, cardiac dyspnea is fundamentally due to disturbances in hematosis produced by congested lung. secondly, in an inconstant manner, other less important factors that we will analyze later, under the name of secondary factors, can also influence its production mechanism, such as:

a) The physicochemical composition of the blood (oxygen, carbon dioxide and hydrogen content).
b) The decrease in cardiac output with its poor irrigation of the respiratory center.
c) Reflex factors through the vagus, the engorged lung, the distended vena cava, the carotid sinus and the birth of the aorta.
d) The increase in oxygen consumption and basal metabolism by the respiratory muscles and by the hypertrophic heart.
e) The decrease in pulmonary blood flow in certain congenital heart diseases and in acute cor pulmonale.

Primary factor of cardiac dyspnea Pulmonary factor

It is due, as we have pointed out, to the alterations in hematosis produced by the congested lung, that is, by pulmonary congestion. We must remember some physiological considerations that are fundamental for the interpretation of cardiac dyspnea. Firstly, let us point out that the amount of air breathed in the unit of time is called respiratory requirement; This is measured in basal conditions, and that respiratory requirement in basal conditions that the subject needs to carry out his natural respiration and normal hematosis is called respiratory requirement at rest. When this requirement is measured during exercise, it is called exercise respiratory requirement. Another fact that we must remember is what is known as maximum voluntary ventilation, which is the maximum volume of air that a subject can breathe, in the unit of time, of their own will, executing forced breaths.

If we subtract the respiratory requirement at rest from the maximum voluntary ventilation, we will obtain what is called respiratory reserve at rest, and if we subtract the respiratory requirement during exercise from the maximum voluntary ventilation, we will find what is called the respiratory reserve during exercise.

These respiratory reserves at rest and during exercise, which are precisely the volume of air that the body can have available once the respiratory requirement has been satisfied, either at rest or during exercise, are altered in cardiac individuals and the physiological state can vary greatly, according to the different subjects. Therefore, its value is not advisable to retain or remember, because the individual variations are quite large. But if we make an index by dividing the

respiratory reserve at rest due to maximum voluntary ventilation and also respiratory reserve during exercise due to maximum voluntary ventilation. These indices at rest and during exercise are extraordinarily fixed and constant in their figures. It can be considered that the index at rest must always be greater than 90% and the index during exercise must always be greater than 72%.

If there is an index at rest less than 90%, or less than 72% during exercise, there will always be dyspnea, the more intense the more any of these indexes decrease.

On the other hand, the respiratory functional unit has two objectives:

1. Maintain gas exchange.
2. Maintain fluid exchange with the pulmonary interstitium.

Functionally three situations can happen:

a) Poorly ventilated and well perfused alveolus: bronchial asthma.

b) Well-ventilated and poorly perfused alveolus: pulmonary thromboembolism.

c) Alveolus well ventilated, well perfused and with poor gas diffusion: respiratory distress.

This respiratory distress or progressive respiratory insufficiency has its basis in the aforementioned pulmonary interstitium, a site where the lymphatic and venous capillaries maintain liquid balance against the alveolar wall.

If this mechanism fails, fluid accumulates in the interstitium and is therefore called the interstitial edema phase of heart failure, whose radiographic expression is the horizontal Kerley lines.

As the fluid is in the interstitium, mainly due to pulmonary venous hypertension, and not in the alveolus, this edema is not accompanied by pulmonary crackles. Therefore, it is not auscultable, given the difficulty in the diffusion of gases and especially Oxygen. This respiratory distress is common to many pathologies. Therefore, depending on the intensity of the interstitial edema, we will see progressive types of dyspnea in the cardiac system. If the installation of interstitial edema is sudden, acute lung edema may occur.

In these cases, water passes into the alveolus, and the crackle and subcrackles are auscultable in this phase.
These moist rales can take the form of "rising tide", that is, they are heard at increasingly higher levels.

The congested lung gives rise, therefore, to pulmonary hyperventilation, that is, to dyspnea due to a decrease in vital capacity as a defense of the organism to produce an increase in hematosis and normal oxygenation.

In addition to congestive lung, pleural effusions, pulmonary infarctions, increased abdominal pressure with elevation of the diaphragm, as seen in ascites, pregnancy, hepatomegaly, etc., and pericardial effusions that compress the lung, especially in children, also contribute, on some occasions, to this dyspnea. In these cases, any of these contributing factors, which are relatively common in cardiac patients, help or contribute together with the congestive lung to produce a decrease in vital capacity. In congestive lung, the fundamental thing is the existence of congestion and increased pressure in the capillaries and pulmonary veins, which gives rise to:

- Decreased capacity of the alveoli.
- Lung rigidity.
- Decreased alveolar permeability.
- Reflex bronchospasm.

We have already said that in congestive lung the fundamental thing is the congestion of the veins and capillaries; There is venous hypertension at the level of the pulmonary veins and capillaries, and to a certain extent in the arterioles. We must now analyze these mechanisms in order to explain why this engorgement and increase in pressure in the veins and pulmonary capillaries can lead to what is precisely called a decrease in lung capacity or vital capacity.

Decreased capacity of the alveoli. The alveolar capacity is decreased because the capillaries that surround the alveoli dilate and become herniated within their lumen; Therefore, these alveolar spaces are also more or less filled with edema fluid that has transuded due to increased venous tension and alteration of the capillary wall.

Pulmonary rigidity: The amount of blood accumulated within the lungs keeps them in a position of inspiration and, therefore, with a predominance of the Hering-Breuer reflex, giving rise to early expiration, which causes breathing to be done superficially and with a very short inspiration.

Decreased alveolar permeability. We also point out that the decrease in the permeability of the alveolar walls results in greater difficulty in the gas exchange of oxygen and carbon dioxide at the level of the lung, which partly makes hematosis (diffusive insufficiency) more difficult.

Reflex bronchospasm. It has been demonstrated through pneumograms obtained from patients with heart failure in which the expiratory phase is lengthened and deformed,

the layout becoming more convex in that part, as observed in allergic asthma. These anomalies have been observed in cardiac patients, whether or not they have paroxysmal dyspnea; So it can be inferred that perhaps in the cardiac case there is a certain degree of reflex bronchospasm due to the pulmonary congestion itself, which would further reduce the vital capacity of these subjects, since, in these cases, and by performing serial pneumograms, it has been seen that the action of aminophylline decreases the expiratory phase and improves the vital capacity of these patients.

On the other hand, pulmonary engorgement is also capable of increasing the frequency of respiratory movements and producing cardiac dyspnea by a reflex mechanism that acts on the respiratory center. We know that under normal conditions respiratory regulation is carried out through the Hering-Breuer reflex mechanism, through which inspiration is interrupted by an inhibitory vagal impulse that originates from the alveoli when their distension reaches certain proportions, then expiration begins, which, in turn, is suspended to reinstate the next inspiration, as soon as the alveoli experience a certain degree of collapse.

The respiratory center is located in the medulla, below the floor of the fourth ventricle. Most researchers distinguish an inspiratory portion in the ventral part of the reticular substance, and an expiratory portion that occupies the dorsal part, although some others accept the point of view that the neurons that constitute the inspiratory and expiratory centers are intimately intermixed, and that the morphological segregation of the two centers is artificial. Harrison's studies demonstrate that the decrease in vital capacity is capable of exciting the respiratory center by a nervous reflex established through the vagus, since in his experiments, carried out on dogs, he found that the reduction of vital capacity through pneumothorax and introduction of liquid into the lung or engorgement of the capillaries of the lung, produces tachypnea, provided that the vagus nerves are preserved intact, while, The same maneuvers, carried out in previously vaguectomized animals, do not accelerate respiratory movements.

tories.

Secondary factors

Let us now consider the secondary factors of cardiac dyspnea:

- Disturbances of the physicochemical composition of the blood.

Decrease in cardiac output with poor irrigation of the respiratory center.

- Reflex factors.

Disturbances of the physicochemical composition of the Blood

In the past, it was considered, according to experiences, that there was a decrease in oxygen, an increase in carbon dioxide and acidosis in heart failure, and that these factors were mainly responsible for dyspnea in cardiac patients.

We will only object that modern research has shown:

1. That these previous studies had been carried out on venous blood and the blood that reaches the respiratory center is arterial blood.
2. That except in advanced cases of circulatory insufficiency, especially right, in which alterations of the components that we have previously indicated are evidently established, these alterations have not been verified except in special cases.

In summary, without discussing all the points of view for and against this criterion, let's say that physicochemical studies of arterial blood prove: anoxemia inconstantly, exceptionally excess carbon dioxide (paCO, high or hypercapnia) and hydrogen ions, so it can be concluded that these factors rarely constitute the primary factor in the mechanism of cardiac dyspnea and only play the role of cause. adjuvant.

On occasions, and when heart failure is advanced and of a congestive type, they can acquire preponderance as a late phenomenon superimposed on the pulmonary factor, as is also seen in some types of congenital heart disease.

Decreased cardiac output with poor irrigation of the respiratory center

From this point of view, it would constitute a symptom of insufficient flow. It has only been possible to find this in cases in which there are advanced vascular sclerosis, which, in turn, disrupt the circulation of the respiratory center.

This mechanism could also be considered in some cases of shock. We know that the brain centers are the last to suffer, because the peripheral vasoconstriction of the limbs and organs forces the blood to circulate in greater proportion through the brain, which is the noblest; However, in advanced cases some polypnea may occur due to this mechanism.

Reflex factors

Reflex factors such as those coming from the vagus, the vena cava, the carotid sinus, etc. are also considered.

It is believed, and at this point there are many confirmations, that it is through the nerve endings, excited by physicochemical alterations of oxygen, carbon dioxide or pH, that polypnea can be established.

In these cases, there is an increase in oxygen consumption and basal metabolism, due, on the one hand, to the greater and more frequent contraction of the respiratory muscles and to the same myocardial hypertrophy. Some assume that this increase in metabolism and oxygen consumption found in cardiac patients would justify dyspnea to try to get enough oxygen to the respiratory and cardiac muscles. The last factor to consider is the decrease in pulmonary blood flow in some congenital heart diseases and in acute cor pulmonale, where the sudden decrease in blood flow in acute cor pulmonale, or progressive in some congenital heart diseases, leads, as we have pointed out, to a type of dyspnea with that special squatting position of patients described by Dr. Taussig.
precisely in patients with decreased pulmonary blood flow.

In summary, cardiac dyspnea is due to a primary factor, which is lung congestion, that is, venous and capillary hypertension with engorgement of the veins and capillaries at the level of the lung, caused by heart failure and edema of the pulmonary interstitium; Added to the congestion are other contributing factors that further reduce vital capacity, such as strokes, infarctions, elevations of the diaphragm or increased abdominal pressure, or pericardial effusions with lung compressions. Also influencing, on the other hand, are the so-called secondary factors, among which are disturbances in the physicochemical composition of the blood, decreased blood flow to the respiratory center, nervous reflex factors, through which many of these alterations act, and decreased pulmonary blood flow in congenital heart disease and acute cor pulmonale. All of them, in a primary or secondary form, and even taking on the character of primary at certain times or in certain types of heart disease, influence to a greater or lesser degree the production or increase in the intensity of dyspnea in the heart.

Classification

Dyspnea in cardiac patients has certain characteristics that we will describe. In its study we must consider five fundamental types:

- Dyspnea on exertion.
- Recumbent dyspnea, of which orthopnea is a modality in its most advanced forms.
- Paroxysmal dyspnea, which in turn has two clinical forms of presentation: cardiac pseudoasthma and acute lung edema.
- Continuous dyspnea.
- Respiratory arrhythmias.

Dyspnea on exertion

Concept and semiography

Exertional dyspnea that occurs, as its name indicates, with large, medium and small efforts, has been called precisely that way because it disappears when the subject is at rest. Depending on the magnitude of the physical effort that produces it, dyspnea of great effort or stair dyspnea is distinguished, which sometimes appears even in any untrained subject where an exercise, perhaps not very intense, can be disproportionately great for him and produce this dyspnea; Sometimes intercourse can trigger it.

Dyspnea with medium exertion appears when minor exercises, such as walking one or two blocks at a normal speed or pace, or slowly climbing a few steps, trigger it.

Dyspnea on minor exertion, which appears during minor exercises, such as, for example, changing posture, the act of dressing, speaking, laughing, etc.

Of course, these degrees of intensity of dyspnea, in turn, mean degrees of intensity of the causal lesion, specifically central circulatory insufficiency.

Taussig has described a peculiar form of exertional dyspnea in patients who have certain congenital malformations, which result in a reduction in pulmonary blood flow or output, such as stenosis, atresia of the pulmonary or tricuspid artery and tetralogy of Fallot, that is, those patients who present congenital malformations such as those already described, which reduce pulmonary blood output, present a special type of exertional dyspnea when They perform exercises, for example, and are forced, to get rid of dyspnea, to adopt the squatting position, and only in this position does the dyspnea of these patients improve.

The explanation for this fact is unknown; However, it tends to be accepted that this posture can produce a certain expression of the engorged abdominal organs, which would increase the blood flow that flows to the right heart and in this way the tricuspid or pulmonary barrier could be strained somewhat, then increasing the amount (debit) of blood that reaches the pulmonary artery and the lung. The truth is that this peculiarity of dyspnea is exclusive to the patients we have mentioned and is not present in any of the congenital cardiac types whose pulmonary circulation is not decreased.

True exertional dyspnea is proportional in its intensity to the intensity of physical exercise, that is, the greater the exercise, the greater the dyspnea; This, however, does not occur in some nervous patients who report dyspnea on exertion with small exercises and yet sometimes do not present it with larger exercises. This subjective dyspnea when starting exercise, which has been called nervous dyspnea or " first effort" dyspnea by Chávez, is typical of subjects who have a neurovegetative or psychoneurovegetative alteration. Most of the time, these patients report their respiratory disturbance to the doctor as shortness of breath, which is more apparent to them when their attention is not occupied; Thus, it is common for them to present it when they have finished their daily shift or during the night (while awake) and it is rare for it to occur during the day while they are carrying out their work. This dyspnea can take two fundamental types in nervous subjects, which have been called gasping dyspnea, because patients say it presents itself in a gasping form, and sighing-type dyspnea, in which very deep breaths are produced that the patient considers himself forced to take. Both sighing dyspnea and gasping dyspnea can lead, if they last for a long period of time, to pulmonary hyperventilation syndrome, which is then accompanied by the symptomatic picture of respiratory alkalosis (hypocapnia), presenting vertiginous sensations, paresthesias, cramps, convulsions and even true tetany on some occasions. It is of great importance for the clinician to differentiate these forms of nervous dyspnea, of the patient affected by circulatory asthenia, from the true exertional dyspnea of cardiac insufficiency, for which the verification of the existence of other symptoms of heart failure that are naturally lacking in these patients with neurocirculatory asthenia helps greatly.

Recumbent dyspnea

Concept

In general terms, recumbent dyspnea is that which appears or is exaggerated when the patient lies down, and decreases or disappears in the upright position of the trunk and head; Precisely because it is relieved by orthostatism, it is also called orthopnea. We must point out that there is a type of dyspnea that has been called recumbent dyspnea, in which the patient has dyspnea after a few minutes of lying down but it disappears a few minutes later, that is, they present dyspnea when lying down and it improves when a few minutes have passed after being in that position, and they can remain lying down; and intense recumbent dyspnea called orthopnea, in which the patient cannot support the recumbent position for a longer or shorter period of time.

and he is forced to sit or stand to rest his hands on some firm object, in this way he immobilizes his shoulders and can use the accessory muscles of respiration to breathe.

Semiogenesis or pathophysiology

Different factors influence the semiogenesis of cardiac orthopnea:

1. The respiratory mechanism of inspiration and expiration, that is, the expansion and retraction of the thorax, is facilitated in the upright position and is difficult when lying down.
   It has been shown that recumbency reduces vital capacity by 6% in normal individuals, and in cardiac individuals this reduction reaches 27%.
2. Pulmonary congestion increases with recumbency and decreases with upright position; This is due to the greater volume of blood that accumulates in the abdominal vessels and in the lower limbs and which has been calculated to be approximately one liter when the subject is in orthostatism; There is also a longer blood circulation time in the lower limbs when the subject is in that position.

3. It has been pointed out that recumbency hinders blood emptying of the cephalic extremity, which gravity helps in orthostatism; This would result in a greater degree of cephalic venous stasis and, therefore, a greater degree of stasis in the bulbar respiratory center; This would explain the clinical fact observed by Blungar, that some patients with mild recumbent dyspnea are relieved by simply raising the head by flexing it on the trunk, without having to resort to the erect position.

We have already pointed out that studies dosing oxygen and carbon dioxide in the blood of the internal jugular and carotid arteries in clinostatism and orthostatism have not shown appreciable differences to support this idea or this hypothesis, nor have they been able to produce dyspnea by moderately compressing the neck and thus increasing cephalic venous pressure. Therefore, the first two factors mentioned seem to be of much greater importance than the last.

Paroxysmal dyspnea

Concept

It consists of attacks of sudden onset of more or less intense shortness of breath, almost always occurring at night, and which are not motivated by physical exertion. Paroxysmal dyspnea adopts two different modalities that

Are:

a) Cardiac pseudoasthma.

b) Acute edema of the lung.

Although from a clinical point of view these two forms can be distinguished, from the point of view of their etiology and semiogenesis they are practically two different modalities of the same phenomenon. In short, acute lung edema is a more advanced degree of cardiac pseudoasthma.

A. Cardiac pseudoasthma.

Semiography:

The intense attack of cardiac asthma is impressive: The patient, frequently during the night, already asleep or in the process of falling asleep, wakes up with intense dyspnea and an anxious state, sits up in bed and holds tightly to the edge or bars of his bed, in order to use the accessory muscles of respiration. The frequency of breathing increases and although it is almost always silent, sometimes it is noisy at a distance, as in bronchial asthma. The patient's expression is one of marked anguish, with his eyes open and exorbitant, his face pale, grayish or cyanotic, sweaty and cold. Cough is common, not intense, dry or with little viscous expectoration, sometimes bloody.

Physical examination of the chest reveals, in severe cases, the polypnea described and the chest in permanent inspiration. The loudness of percussion is often increased, although it may be normal or decreased.

especially in the infrascapular regions.

Auscultation may reveal the presence of wheezing, subcrackling, or crackling rales; the latter, the more numerous the more pronounced the alveolar edema, which is always present to a greater or lesser degree.

B. Acute edema of the lung.

Semiography:

This dyspneic condition may appear in the course of prolonged cardiac asthma, or flare up with the characteristics of acute pulmonary edema from the onset of the dyspneic episode. It is very similar in its clinical appearance to the attack of cardiac pseudoasthma, but, generally, the dyspnea is more intense, truly suffocating breathing, with a sensation of constriction in the chest or neck. The expectoration is abundant, fluid, foamy and with a greater or lesser amount of blood, depending on its color, from yellowish white to pinkish, rather salmon-coloured, or bright red. Due to its consistency and foamy appearance, it resembles poorly beaten egg white. It is also common for the outbreak of acute edema to begin with a sensation of pharyngeal tickling caused by the presence of edema fluid in the upper parts of the respiratory tree. The patient's attitude is the same as that of those who suffer from cardiac asthma, as well as the time and circumstances in which it appears; cyanosis or shock is more frequent and more intense; The moist rales take the form of "rising tide" already described.

Semiogenesis or pathophysiology:

In the semiogenesis of paroxysmal dyspnea, whether cardiac pseudoasthma or acute pulmonary edema, the mechanical factor has long been considered responsible, that is, this dyspnea is attributed to a functional deficit of the left ventricle; However, although there are numerous arguments in favor of this hypothesis, this mechanical theory of the determination of dyspnea, so suggestive for the clinician, as it fully adapts to most of the facts observed in the domain that belongs to it, is, however, incompletely explained and is sometimes in clear contradiction with other observations in man and in experiments on animals. This is how the following facts cannot be explained by mechanical theory:

1. Exceptional but obvious cases of acute pulmonary edema in patients with lesions of the nervous system without cardiac alterations (for example: head trauma, brain tumors, encephalitis, etc.), during acute pulmonary infections (such as pneumonia) or pulmonary infarctions, or poisoning by gases such as phosgene.

2. The surprising action of the injection of morphine to jugulate the accidents of paroxysmal dyspnea and the favorable effect exerted by this and other hypnotics, to prevent the installation of acute edema experimentally provoked in the dog by injection, or rather massive infusion into the carotid, of a saline solution. Furthermore, it has been shown that this continuous saline infusion into the dog's carotid, which inevitably leads to acute pulmonary edema, does not do so when the infusion is carried out in peripheral arteries other than the carotid, which suggests that there is a central cephalic factor that plays a great role. At the same time, acute lung edema crises have been produced experimentally through bilateral vagotomy, or with the injection of a high dose of adrenaline.

The insufficiency, therefore, of the mechanical theory to explain the above facts, justifies the neurogenic theory, which admirably completes the mechanical concept and in favor of which Luisada has contributed his valuable experimental studies.

On the other hand, there are some arguments that we must also point out, such as paroxysmal dyspnea motivated by emotions, and also the proven fact of the importance of other reflexes with different starting points in the production of acute pulmonary edema, such as reflexes that originate from the atrial and ventricular walls, the aorta, the superior cava or the pulmonary veins themselves.

In summary, in the semiogenesis of paroxysmal dyspnea it can be assured that the fundamental cause that produces the crisis of paroxysmal dyspnea lies in sudden pulmonary congestion and respiratory distress caused by the functional deficit of the left ventricle or by mnitral stenosis, but to this mechanical factor other factors are added to a greater or lesser degree such as the active dilation of the pulmonary capillaries, whose permeability increases due to the excitation of nervous elements due to different reflexes. These reflexes can arise in the heart itself (for example, in myocardial infarction, myocardial infections or degeneration, sudden myocardial distention), or in the carotid sinus and aorta (in hypertensive outbreaks), or in the superior cava (congestive heart failure), or in the engorged thrombosed pulmonary vessels themselves, and even in the cerebral cortex itself from an emotional point of view.

Continuous dyspnea

Concept

As its name indicates, it is permanent; It is observed in patients who present not only passive visceral congestion, but also edema that reaches the maximum degree of anasarca, sometimes including ascites, caused by severe congestive heart failure in the terminal stage.

Respiratory arrhythmias

The most frequent are:

a) Cheyne-Stokes breathing.
b) Kussmaul breathing.
c) Biot Breathing.

Of these, the one that is of interest in the cardiac system is Cheyne-Stokes dyspnea, which we will not go into describing, as it has been completely studied in the respiratory system.

PALPITATIONS

Concept

Palpitation is the conscious perception of cardiac activity, in an annoying way, due to the stimulation of its somatic, tactile and baresthetic sensitivity, by the translational, vibratory and expansion movements that They occur during each cardiac cycle. It can appear physiologically, this is the case of the individual who cannot do violent exercises because he feels the shock of his heart, or it can appear in pathological conditions, both in the course of cardiac and extracardiac conditions.

Semiognesis or pathophysiology

Normal cardiac contraction is not usually perceived due to a phenomenon of habituation or fatigue of the nerve endings that are excited with each contraction; Therefore, for this to occur, a stimulus of different quality and intensity is necessary, or one that occurs at the wrong time, or in a different way than usual.

When the nervous system is hyperexcited, normal contractions can cause the same phenomenon.

less

semiography

If the stimulation occurs in the structures adjacent to the heart, the palpitation will be felt in the precordial region, in its vicinity, or in both: cardiac palpitations. If the result of the expansive movement of the arterial or venous system, due to the transmission of the pressure wave produced in the systole of the left ventricle, will be felt far from the heart, being called by some: vascular palpitation; It's actually more of a heartbeat.

heart palpitations

Some authors divide them into:

- Regular palpitation.
- Irregular palpitation.

regular patpitation

It is one in which cardiac activity is perceived regularly, but more accelerated: tachycardia.

Irregular palpitation

In it, cardiac activity is perceived in an irregular, disordered and non-rhythmic manner: extrasystole, arrhythmia, etc.

According to their periodicity, palpitations are classified as:

- Isolated
- Grouped or grouped.

Isolated palpitations

They are perceived as a jerk, hit, jump, overturn or failure. All these sensations are of very short duration, less than a second, instantaneous, and several can be repeated in a minute or after periods of more or less long time.

Sometimes they are associated with a cough or a short-lived feeling of dizziness or obtundation, which does not give the patient time to become alarmed.

They constitute the subjective sensation of an extrasystole.

This, as we know, is nothing more than a premature systole followed by a compensatory pause.

This anticipatory excitation can be generated in the atrium or in the ventricle and, therefore, there will be atrial extrasystoles and ventricular extrasystoles.

Ventricular extrasystoles. They appear earlier, therefore, in them the compensatory pause will be greater.

This pause is nothing other than a lengthening of diastole that causes greater ventricular filling and causes a more energetic following systole, so that the pause is nothing other than a lengthening of diastole that causes greater ventricular filling and consequently causes a more energetic following systole. The patient may perceive premature systole as an overturning of the heart, or as a pinch or puncture. This discomfort is followed by the sensation of cardiac arrest or arrest, which is frequently accompanied by anxiety or fear and which corresponds to the compensatory pause that follows the extrasystole. After it, the patient can perceive an energetic heartbeat, a true water hammer, simultaneous with the post-extrasystolic contraction.

There are patients who clearly notice this whole set of sensations, derived respectively from premature systole or extrasystole, from the pause that follows it and from the ventricular contraction that resumes sinus rhythm; but others only perceive one or two of these successive phenomena, most commonly the pause and the turn, sometimes none of them. The frequency with which premature systoles appear and the regular or irregular succession with which they occur make the sensations that the patient can notice vary infinitely.

Semidiagnosis:

Extrasystoles can be due to different causes (fig. 37.3):

1. Extracardiac causes.

2. Cardiac causes.

1. Extracardiac causes.

Extrasystoles are seen with great frequency in:

a) All digestive disorders such as gastric dyspepsia, aerophagia, colitis and cholecystopathies.

b) Genital disorders in women.

Its mechanism is therefore due to a reflex cause of neurovegetative origin.

2. Cardiac causes.

They can produce or frequently produce extrasystoles:

a) Valvulopathies.

b) Heart failure, in which these extrasystoles accompany dyspnea on exertion and therefore have the same significance.

c) The anxiety neuroses that appear in the so-called soldier's heart or irritable heart, a clinical condition that presents variable symptoms and in which extrasystoles are extraordinarily frequent.

Grouped or grouped palpitations

They are successive sensations of blows or beats, grouped together in a burst or flutter, as their name indicates, with accelerated frequency, and can last from seconds to hours and even days, or be permanent; They accompany organic pathologies or previous cardiac functional alteration.

There are two different modalities, each with a different meaning:

- Type I.
- Type II.

Type I. Gradual or abrupt beginning and always gradual ending. These palpitations occur practically only during wakefulness; They can be episodic or permanent, spontaneous or caused by physical efforts or emotions. They invariably have a regular rhythm and accelerated frequency, but never higher than 150/min, except in infants or early childhood, which may be higher.

Semidiagnosis:

1. In healthy individuals in situations of great emotion or unusual physical effort, or in hypersensitive subjects when lying on the left side.
2. In neurocirculatory asthenia or psychoneurosis.
3. In hyperthyroidism, caused by the sympathicotonia that accompanies it.
4. In anemia.
5. In the narrowing or stenosis of the mitral valve.

Type II. They always have abrupt beginnings and endings.

They can occur both during wakefulness and in full sleep, for brief or prolonged episodes and even permanently. They generally appear spontaneously, but they can be triggered by efforts or emotions, and their frequency is almost always greater than 160 beats/min.

A STADOD ANS DAD Palpitations may accompany rare episodes of acute anxiety or occur frequently, accompanied by chest pain and dyspnea

B M D CAM NTS These include nicotine, caffeine, epinephrine, ephedrine, atropine, aminophylline, alcohol, and thyroid extracts.

CT ROT0 COS S When this is complicated With heart failure, the cause of palpitations is usually obscure unless thyroid function tests are

D PROC SOS F BRL S Palpitations may be a prominent symptom in early pulmonary tuberculosis, pneumonia, and brucellosis, and a late manifestation in chronic glomerulonephritis.

CONDITION DISORDERS CC N Intrinsic cardiac disorders, such as coronary artery disease and rheumatic myocarditis or myocarditis secondary to bacterial endocarditis, are capable of causing

FT MOR SS PRARR NAL S Iparaganglioma (pheochromocytoma) of the adrenal medulla causes hypertension and bouts of palpitations, as usually observed consecutively when injecting epinephrine

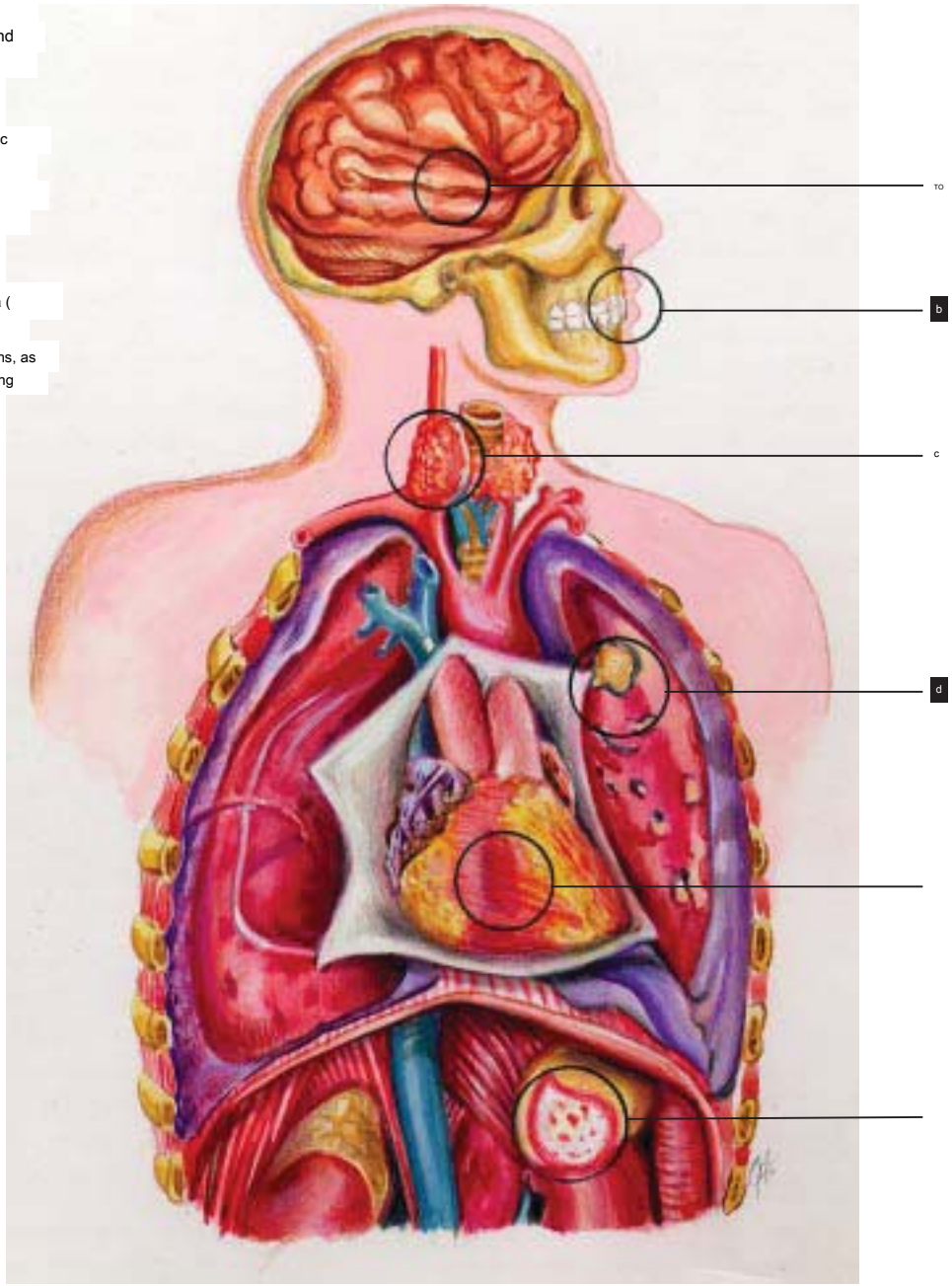


Fig. 37.3 Diseases that most frequently cause palpitations.

Semidiagnosis:

Grouped or burst palpitations can be:

1. Rhythmic:

a) In paroxysmal tachycardia.

b) In paroxysmal atrial flutter.

2. Arrhythmic:

a) In paroxysmal atrial fibrillation.

Paroxysmal tachycardia is a successive uninterrupted group of extrasystoles. In this case, an abnormally excitable area of the heart assumes control of this organ, giving rise to a series of rapid heartbeats.

The symptoms that the subject suffers from are the following: an isolated knock or palpitation followed by a perceptible rapid activity that lasts minutes, hours or days and that ends abruptly as it began or sometimes with a sensation of precordial shock.

Atrial flutter is characterized by the fact that its contractions are more frequent in the atria than in the ventricles, at a rate of 2:1 or 4:1, causing burst palpitations that are also irregular in appearance.

In paroxysmal atrial fibrillation there is an alteration in the way atrial stimuli are conducted; In this case, the entire atrial wall is excited, generating circles around the atrium that can be followed or not by ventricular contractions, therefore the ventricular contractions will be unequally separated in intensity, in time of appearance, etc. Before permanent fibrillation is established, paroxysmal fibrillation will cause rapid palpitations of irregular type and character. Fibrillation is also called delirium cordis or madness of the heart, complete arrhythmia or perpetual arrhythmia.

vascular palpitation

It corresponds to the arterial beat; It is generally a sensation of an isolated or successive heartbeat, with regular or irregular frequency and cadence depending on the heart rhythm.

The arterial beat can be perceived in any part of the body, as long as there is a wide differential pressure as in aortic insufficiency, hyperthyroidism, etc., and also when the semi-crushing of an artery of a certain caliber occurs due to extrinsic compression, for this reason the selective sites to be perceived are the limbs and the head in certain positions or when they are supported on hard planes. It can also be perceived in the active dilation of muscular arteries (throbbing headache in migraine), in sites corresponding to acute inflammation (paradentary phlegmons or subcutaneous phlegmons) and in arterial aneurysms.

Venous palpitation, due to a retrograde systolic wave towards the venous system, can be seen in the presence of tricuspid insufficiency, ventricular tamponade in patients with nodal rhythm, supraventricular tachycardia in which the systole of the ventricles coincides with that of the atria, in these cases the location of the beats is at the base of the neck (positive venous pulse) or in the epigastrium (positive hepatic positive).

ENCEPHIC CIRCULATORY MANIFESTATIONS

Vertigo and seizures are seen more often in conditions other than the cardiovascular system. On the other hand, lipothymias and especially syncope often constitute a manifestation of cardiac or arterial suffering.

The lack of blood supply to the brain gives rise to brain ischemia, which causes vertigo, lipothymia, syncope and convulsive attacks, depending on the duration of the ischemia.

If the ischemia lasts a few seconds, vertigo and lipothymia occur; If it lasts a little longer, syncope and convulsions appear.

vertigo

Concept and semiography

Vertigo is an unpleasant sensation of instability produced by a disorder in the sense of balance. The subject with vertigo mistakenly feels that his body rotates around or in the middle of the objects that surround him (subjective vertigo), or that the objects that surround him rotate around him (objective vertigo), and sometimes both at the same time. As a result of this, the patient may falter or fall when standing or while walking. It is associated with a feeling of anxiety or fear, and may be accompanied by a state of nausea, vomiting, ringing in the ear, general malaise, paleness and sweating.

Vertigo in cardiovascular conditions

Vertigo is not a common symptom in cardiovascular conditions, however, it is of great importance in conditions of the ear (labyrinth) or the central nervous system.

Any pathology located in the balance pathways or centers can cause vertigo. Therefore, we will study it in detail in neurological semiology.

Semidiagnosis

We will only point out here that disturbances in brain circulation can cause vertigo. Therefore it can be seen in:

1. High blood pressure.
2. Cerebral atherosclerosis.
3. Arterial hypotension due to aortic stenosis or insufficiency.
4. Myocardial infarction.
5. Paroxysmal tachycardia.
6. Shock.
7. Orthostatic arterial hypotension observed in hypertensive patients treated with ganglionic blockers.
8. Hypersensitivity of the carotid sinus.
9. Complete atrioventricular block.
10. Cyanosis or anemia.

Lipothymia

Concept and semiography

Also called by the sick fainting, dizziness or fainting; It consists of the temporary clouding of consciousness, and therefore of all the senses.

Sometimes the patient experiences only a darkening of vision with muscle weakness, particularly manifested by weakness of the lower limbs, accompanied by nausea, cold sweats, paleness and coldness of the extremities. On other occasions the temporary loss of consciousness is total and causes the patient to fall.

In lipothymia, the persistence of the heartbeat can be verified by palpating the pulse; and if it is very weak, by auscultation of the heart.

If the clouding is very fleeting, it is called absence.

Syncope

Concept and semiography

It is a more pronounced disorder than lipothymia. In syncope, consciousness is completely lost, which causes the patient to fall. Furthermore, the heart stops beating, so that the pulse and heart sounds are completely absent, even though in some cases the electrocardiographic recording taken at that moment allows recognition of some abnormal activity of the heart, such as ventricular fibrillation.

When the systolic ejection disappears, the patient acquires a cadaverous pallor.

If the ventricle does not resume its beating soon, alterations in the functioning of the respiratory center occur due to anoxia; Breathing becomes difficult and the automatism of the center may be lost, which causes the patient's color to transform from pale to cyanotic.

In these cases, as cardiac and respiratory functions are abolished, in addition to cyanosis, lockjaw, abundant foaming of the lips, conjugated deviation of the eyes and convulsions.

Syncope is by definition a temporary alteration, which if prolonged would cause the death of the patient.

After a few seconds or a few minutes the heart resumes its march and each of the aforementioned phenomena quickly dissipates.

Syncopal states can recur in the same subject.

Semiogenesis and semidiagnosis

The initial disturbances that will secondarily cause the series of phenomena that lead to syncope are fundamentally three:

-The sudden reduction in blood flow to the heart determines what some authors call precardiac vascular syncope.
- Heart failure or cardiac syncope.
- The initial cerebral disturbance, which causes cerebral or postcardiac syncope.

Precardiac vascular syncope

It is the most common type, representing 90% of syncope. It occurs, as has been said, due to a sudden reduction in blood flow to the heart, which can result from the following causes:

- Sudden decrease in vascular tone in the abdomen and lower extremities.
- Sudden increase in endothoracic pressure.
- Postural influences.

Sudden decrease in vascular tone in the abdomen and lower extremities. This determines that blood is momentarily stored in these regions, with a consequent reduction in flow to the heart and a drop in blood pressure. In this syncope called vasovagal by Lewis (1932), although it is more appropriate to call it vasodepressor syncope, cerebral flow decreases significantly and the oxygen saturation of the internal jugular vein is between 11 and 24.5%.

Syncope due to this mechanism is common in subjects with neurovegetative dystonia, as well as in young women and people in poor health, in the weakened, anemic, and in those convalescent from different diseases. They occur more easily when the subject is in an erect position.

Vasodepressor syncope can be triggered by different factors, for example:

a) Psychological or sensory stimuli. An unpleasant impression: the sight of blood, a corpse, trauma, etc.; an emotion; unexpected news often causes loss of consciousness.

b) Viscerosomatic stimuli. The excitation of certain organs rich in nerve endings can produce vasodepressor syncope in normal subjects. Engel, Romano and McLin (1944) caused this variety of syncope by venipuncture, or distension of the duodenum, colon, rectum and vagina. Various pathological processes that occur in these organs make them more sensitive. They can also occur in some brain tumors and in cerebral arteriosclerosis.

c) Drugs. Some medications such as amyl nitrite, sodium nitrite, and nitroglycerin can cause syncope when the patient is standing.

Sodium nitrite acts by decreasing venous tone in the splanchnic area, arms and legs, thereby considerably increasing the vascular bed. The heart cannot compensate for the sudden reduction in peripheral resistance by increasing cardiac output, due to decreased blood supply.

d) Pulmonary hyperventilation. Pulmonary hyperventilation causes, after 2-3 minutes, in healthy subjects, a set of symptoms: tingling and a sensation of dullness or emptiness in the head, dizziness, dry mouth, blurred vision of objects and, if the phenomenon is prolonged, tetany. These phenomena are attributable to the decrease in CO tension in arterial blood. The hypocapnia caused in this way usually causes a moderate decrease in systolic and diastolic blood pressure and a reduction in cerebral flow. However, it is generally accepted that hyperventilation alone does not cause loss of consciousness, except in exceptional cases, which would be caused by the interaction of one of the following mechanisms: orthostatic hypotension and a sudden decrease in vascular tone, due to fear or anxiety of the patient when feeling the discomfort of hyperventilation. Other times the syncope would be of a cerebral type, or of a hysterical nature.

Sudden increase in endothoracic pressure. Some subjects experience sudden loss of consciousness due to the simple act of coughing. These are, in general, subjects with a picnic habit, with a prominent abdomen, good eaters and drinkers, who have passed the middle age of life, or subjects who suffer from chronic lung conditions.

Charcot (1876) was the first to describe this syncope and called it "laryngeal vertigo." The loss of consciousness occurs immediately after the coughing fit has ceased and is due to the fact that during the fit it is difficult to fill the heart by considerably increasing the endothoracic pressure, which is why the output cardiac decreases a lot; consequently, blood pressure drops. Loss of consciousness appears when the pressure reaches values below 50 mm Hg.

Postural influences. When a healthy person is immobile in a standing position for a long time, blood accumulates in the lower extremities and abdomen, reducing flow to the heart. It has been calculated that under these conditions around 500 mL can be stored in the lower extremities and that cardiac output decreases by 5%, having been proven to increase venous pressure in the legs and decrease the filling pressure of the heart.

These alterations can determine the loss of consciousness.

This variety of syncope is common in soldiers standing at attention and in large crowds.
It appears more easily in hot environments or after great efforts and in tired subjects.

Other times the syncopal reaction or syncope in a standing position, or due to standing up abruptly, represents a symptom of a general condition. They are usually observed in weakened subjects, in anemic individuals, in acute hemorrhages and in the convalescence of numerous diseases, and after the administration of hypotensive medications, where they appear temporarily.

There is a more serious variety of syncope which has been called chronic orthostatic hypotension or postural hypotension in which this imbalance is persistent and occurs every time the subject wants to stand up.
It can be observed in conditions of the central nervous system (tabes, syringomyelia), in Addison's disease, etc.

cardiac syncope

This syncope is the most common after vasodepressor syncope. It is usually caused by a cardiac disorder that significantly reduces minute volume. The most common causes are the following:

- Cardiac arrest.
- Extreme sinus bradycardia.
- Ventricular fibrillation.
- Paroxysmal atrioventricular block.
- Sudden failure of the contractile energy of the heart.

Cadiac unemployment. It generally results in loss of consciousness after 4-8 seconds when the subject is standing, and after 12 seconds when the subject is supine. Seizures usually occur later, 30 seconds after the seizure occurs.

We will mention among the most important those that arise due to psychic stimulation (a great emotion, an unpleasant impression, etc.); due to viscero-somatic stimuli (trauma to the genital organs, larynx, epigastrium, etc.): due to intense pain; by swallowing (Flaum and Klima, 1933): by dental extractions; during bronchoscopy; by intravenous injection of drugs or other substances, such as those used to perform excretory pyelography.

Cardiac arrest often occurs by a reflex mechanism, as occurs in the vagal type of carotid sinus syndrome and in many surgical syncope.

Extreme sinus bradycardia. This type of bradycardia is caused by vagal irritation.

Ventricular fibrillation: When ventricular fibrillation occurs, the heart stops expelling blood, and loss of consciousness occurs almost immediately.
This arrhythmia can occur in a paroxysmal form and thus be a causative agent of syncope. Other times ventricular fibrillation does not stop spontaneously once it starts, and causes death unless the doctor intervenes in time. This is seen in subjects who drown or those who have suffered accidental electrocution. Ventricular fibrillation has also been confirmed in patients anesthetized with cyclopropane or chloroform;

shortly after an injection of adrenaline; in the course of cardiac catheterization; when doing electrocoagulation in the vicinity of the heart, etc.

Paroxysmal atrioventricular block. In the case of complete paroxysmal atrioventricular block, syncope may occur due to cardiac arrest that occurs at the end of sinus rhythm and before the appearance of idioventricular rhythm.

Sudden failure of the contractile energy of the heart. In some circumstances, cardiac contraction suddenly becomes ineffective. This can occur in acute coronary occlusion, when myocardial nutrition is disturbed.

Other times syncope is caused by exertion in some heart conditions, such as angina, aortic stenosis, and pulmonary hypertension.

It is common in aortic stenosis, where it appears in 25% of cases (Hammarsten, 1951). It is generally associated with anginal pain and can often be avoided if the patient stops the effort. It seems to come from the sudden failure of the contractile energy of the heart caused by myocardial ischemia.

Cerebral or postcardiac syncope

Two varieties must be distinguished:

- Reflex syncope.
- Local disturbance of cerebral irrigation.

Reflex syncope. The classic type of this variety is carotid sinus syndrome. It is characterized by loss of consciousness, which may be associated with paleness or flushing of the face and hyperpnea. Pulse rate and blood pressure are largely unchanged.
The electroencephalogram reveals large waves with a frequency of 2-4 per second. Focal neurological phenomena (paresis, aphasia, localized seizures, etc.) are often observed during the period of unconsciousness and

This syncope is attributed to a reflex disturbance of the brain centers that maintain the conscious state (preferably, those of the hypothalamus). For other authors, it would occur due to ischemia of these centers due to a localized arterial constriction.

Local disturbance of cerebral irrigation. When the arteries that supply the brain (brachiocephalic trunk, carotid, vertebral, circle of Willis, etc.) are obstructed by processes of a diverse nature (arteriosclerosis, etc.), sudden ischemia of the brain and syncope can occur, as a result of physical efforts, or with hyperextension and rotation movements of the head.

This explains the syncope that can occur in disease of the aortic arch arteries (pulseless disease or Takayasu disease), and in occlusive processes of the vertebral arteries.

Compression of one of the carotid arteries can cause sudden cerebral ischemia and syncope, when the caliber of the artery on the other side is greatly reduced.

OTHER SYMPTOMS

In relation to diseases of the cardiovascular system, the interrogation can reveal many other symptoms, which, although not as important as those already described, can be of some use. We will only refer to them briefly.

Isolated cough

Cough with scant or abundant hemoptoic sputum due to passive pulmonary congestion, or with abundant, pink serous sputum due to acute pulmonary edema.

Persistent unproductive cough

Persistent cough, due to vagal excitement, due to pulmonary stasis in acute left ventricular failure, or due to compression of an aortic aneurysm or an atrial enlargement on the trachea or bronchi.

Aphonia or dysphonia

It may be temporary or permanent due to compression of the recurrent nerve. It is transient when it is due to dilation of the pulmonary artery due to passive hypertension, in

those with left heart failure; and permanently, due to aortic arch aneurysm or atrial enlargement.

Dysphagia

It occurs due to esophageal compression caused by sacciform or dissecting aneurysms of the thoracic aorta, or by aortic anomaly, or by abnormal emergence of the left subclavian.

Reflex nausea and vomiting

They occur due to heart attack or congestion of the digestive system.

pathocellular jaundice

It is due to the alteration of hepatocytes in prolonged chronic congestive failure; hemolytic jaundice, recurrent pulmonary infarction.

Oliguria and pola uiuria

It is due to a decrease in renal flow, even though there is good functional capacity in the kidneys.

Fever

It occurs in active rheumatic carditis, bacterial endocarditis, cardiac infarction and pericarditis.

CIRCULATORY SYSTEM. CHANGES IN THE GENERAL EXAM

38

In a general physical examination of the circulatory system we must keep in mind aspects such as: gait, attitude, facies, skin color, petechiae, Osler's nodules, rheumatic nodules, collateral circulation, edema, clubbing fingers or clubbing fingers, constitution and body temperature, which constitute an extraordinary contribution of semiological value.

BRAND

In circulatory conditions, gait can provide some data of diagnostic importance.

Firstly, subjects affected by heart failure present a special gait, a consequence of the disorders they suffer. These patients are almost always edematous, with intense dyspnea that may be due to exertion at first, but when edema appears, it is generally continuous and often with ascites, that is, with a fluid collection due to transudation in the peritoneum.

The march is slow, in small steps; Sometimes the patient has to stop.

Edema of the lower extremities makes walking more difficult and when the subject has ascites, and it is abundant, the appearance is characteristic, as if he were pregnant.

Another gait alteration is that of the patient who has angina pectoris. These subjects, who walk with some care, from step to step, stop when they are attacked by the pain, trying to make it disappear through rest, and they walk again a few steps more or less, depending on the intensity of the process, until another stop.

Also, individuals who have an arterial lesion in one of their lower limbs, whether due to vascular sclerosis or syphilitic arteritis, etc., are forced to adopt a special type of gait, stopping from time to time - which is called intermittent claudication, even though in these cases the painful manifestation is preceded and accompanied by a certain limp.

ATTITUDE

After considering the gait, we proceed to study the attitude of the cardiac patient.

First of all, the attitude of these patients is very characteristic, when due to their heart failure they have dyspnea, and this dyspnea takes the special type that we call recumbent dyspnea, since it appears in the recumbent position and forces them to maintain a more or less erect position (depending on the intensity of the process), which we have studied under the name orthopnea.

There are times when this attitude is transformed a little and it is not just the sitting attitude, but the subject tends to lean forward in a very marked way, practically lying forward, supported by pillows, this attitude being called the pillow sign. It resembles the attitude of Mohammedan prayer. These are almost always individuals who have significant pericardial effusions in whom this attitude, with the trunk forward, reduces the intrathoracic pressure exerted by the pericardial effusion.

The attitude is also very characteristic in individuals who have chest pain, whether it is the rapid pain of angina pectoris, or the long-lasting, intense pain of a heart attack; The attitude of the patients will be different in both cases.

The individual who has an attack of angina itself generally immobilizes himself as completely as possible. This contrasts in part with the relative mobility of the patient who has anginal pain due to a heart attack.

The first knows that the effort causes pain and tries to move as little as possible, hardly breathing, or doing so superficially, so that not even the movement of breathing increases the pain.

The other, which is usually the first time he is attacked by pain, which is very intense and prolonged, almost always appears restless, tends to move, changes position, until the doctor's advice warns him that it is necessary to maintain the most absolute immobilization. They are, therefore, two quite different attitudes.

These examples, which are the most typical, are enough to understand the importance of this exam.

FACIES

In a general physical examination of the circulatory system we must observe the facies, in the physiognomic expression of the subject, which is also an interesting aspect from the point of view of semiological value.

There are some characteristic facies. The facies in valvular lesions offer two types of sufficient contrast so that we can easily distinguish them and give them diagnostic value.

Aortic insufficiency is due to the inability of the aortic sigmoid to occlude the corresponding orifice at the time of diastole; This causes blood to reflux from the aorta to the left ventricle, which in turn creates in the arterial circulation, a sudden decrease in diastolic pressure that is manifested by large oscillations, since the ventricular systole is much more intense because the volume of blood causes the ventricle to dilate and hypertrophy. A very high systolic pressure is thus generated, which gives rise to a large separation between the maximum and minimum pressure, that is, a large differential pressure. In the arteries this causes large arterial beats, and in the capillaries, phenomena known as capillary pulse, by virtue of which the capillaries are filled and emptied with the heartbeats.

These phenomena of congestion and capillary depletion are manifested by alterations in the color of the affected areas. The facies in this condition is generally pale, but this pallor of the aortic patient is immediately confirmed by the arterial jumps that become visible in the temporal and carotid arteries. He is, therefore, a pale subject, with characteristic arterial jumps.

Sometimes it is possible that these capillary pulse phenomena become visible on the cheek, and then redness and paleness are seen alternating and synchronous with the heartbeat. This facies contrasts with that of individuals with mitral valve lesions, whether mitral insufficiency, mitral stricture, or both. Here too the subject is pale, but pale yellowish, and what stands out most is the venous stasis, which is manifested by cyanosis and which occupies the most prominent places, for example, cyanotic rims are observed on the nose, lips and ears.

The existing pulmonary arterial hypertension affects the right ventricle, which in turn causes the right atrial drainage to be insufficient, and, therefore, peripheral stasis becomes visible in all regions, especially in the head, and as a consequence, in addition to cyanosis, venous engorgement is observed:

the veins of the face are dilated.

The facies is also characteristic in other circulatory processes. In angina pectoris, the subject's face has an anxious, distressing expression, which is difficult to describe and is accompanied by great pallor. It is possible to collect data by physiognomic expression, of course, in congenital conditions and other conditions in which cyanosis is very intense. Cyanotic facies will appear in cyanotic heart diseases, such as tetralogy of Fallot and other forms of congenital heart disease, and in mediastinal syndromes with compression of the superior cava, where they are accompanied by collateral venous circulation, cyanosis in slave and edemna in slave.

SKIN COLOR

It is of great importance to pay attention to the color of the skin. First of all, to a type of skin color: cyanosis (bluish and violet color of the skin), whose significance we already studied.

Conditions of the circulatory system are where both peripheral and central cyanosis occurs most frequently, in various forms.

Among the changes in skin color that are of semiological interest and that can be observed in patients with the circulatory system, we have paleness, which we have referred to as the characteristic paleness of the aortic facies and bacterial endocarditis.

Jaundice is another of the skin color disorders that we will find in the course of circulatory processes. It will have two main origins here.

These are frequently blood extravasations caused by pulmonary infarctions. Hemoglobin disintegrates and becomes bile pigment.

There is also the possibility of jaundice of hepatic origin, as a consequence of heart failure, especially failure of the right cavities, causing chronic circulatory stasis, with an increase in the size of the liver. Passive hepatic congestion is thus established, which alters the function of the liver and ends up also altering its texture, leading to the development of fibrous tissue that constitutes a form of cardiac cirrhosis, which can lead to hepatogenic jaundice in the course of this process.

PETE UIAS OSLER'S NODULES AND RHEUMTIC NODULES

The general examination also allows us to collect semiological elements of great diagnostic interest. We will refer to three, especially: petechiae, Osler's nodules and rheumatic nodules. These elements stand out for their close relationship with circulatory processes.

PETE UIAS

Petechiae were already described in the chapter corresponding to skin. We will only insist that it is a valuable diagnostic element, which should be sought especially when infective endocarditis is suspected.

OSLER'S NODULES

Concept

Osler's nodules are small formations the size of a grain of millet or a pinhead, that appear suddenly on the tips of the fingers, in the thenar and hypothenar regions of the hands and less frequently in the fingers and soles of the feet, and in the interdigital membranes.

Semiognesis or pathophysiology

Its production is attributed to local inflammatory, toxic or allergic changes in the wall of the capillaries or arterioles, therefore, these are vascular lesions in which one or more capillaries are occluded by the intense proliferation of the intima accompanied by desquamation, thrombosis and necrosis. There is perivascular inflammation with polymorphonuclear cells and histiocytes, and diffuse cellular inflammation of the dermis, sebaceous glands, and nerve endings. There was the concept, now discarded, that Osler's nodules were due to bacterial microemboli.

semiography

The most outstanding characteristic of Osler's nodules is that they are painful; This pain has a sudden and very sharp onset, sometimes resembling the prick of a needle.

Upon inspection, they are manifested by a papule with a bluish, pink or red tint; Sometimes they have a darker center. They usually last from one to several days, but may disappear in a few hours.

Semidiagnosis

Their clinical value is considerable, since when they are correctly perceived, according to Libman, they are considered a pathognomonic sign of subacute bacterial endocarditis.

RHEUMTIC NODULES

Also called Meynet nodes. They are the size of a grain of rice, sometimes larger. They are observed in the subcutaneous cellular tissue adhered to the underlying layers and are most frequently located on the extension surfaces of the wrist, knee, ankle and elbow. Their consistency is soft and they can disappear after several days, although they usually harden and persist indefinitely. They are found in cases of rheumatic, active and chronic disease.

COLLATERAL CIRCULATION

The general examination provides, as another element of interest, the collateral circulation, which - whether venous or arterial - is important.

In relation to venous collateral circulation, it is enough to know that it will appear wherever there is a difficulty.

local circulatory system, whether in the vein of a limb or in the superior vena cava, in both cases we will find a characteristic collateral circulation that tries to reestablish drainage through the collateral veins.

We can know the direction in which the blood flows by using two fingers to efface the vessel and removing the compression at both ends. Alternatively, we will check in which direction the explored glass is filled.

This collateral venous circulation is not always obvious to the clinician, which is why it is sometimes necessary to resort to special radiological examinations.

In the compression of the superior vena cava, for example, we have three variants depending on the site of compression:

a) Compression above the mouth of the azygos (anazygotic cava-cava type). The current is reversed in the subclavian veins, and through the internal mammary and costoaxillary veins it pours into the azygos and through it returns to the superior vena cava (Fig. 38.1l).

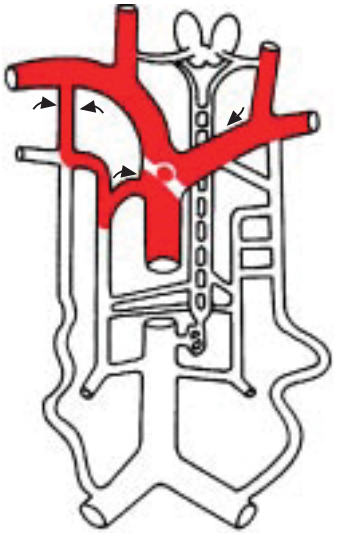
In these cases the collateral circulation is not very marked in the anterosuperior part of the thorax.

b) Compression at the mouth of the azygos.

The blood that comes through the superior cava, unable to drain into the azygos, uses a rich subcutaneous anastomotic network in the thorax and abdomen, dependent on the external mammary, external circumflex and thoracohypogastric. The collateral circulation is very obvious (fig. 38.2).

c) Compression below the mouth of the azygos. Here the circulation is through the venous system

Fig. 38.1 Obstruction of the superior cava above the mouth of the azygos vein: 1, thoracodorsal vein;
2, trunk of the superior intercostal veins; 3, azygos vein.



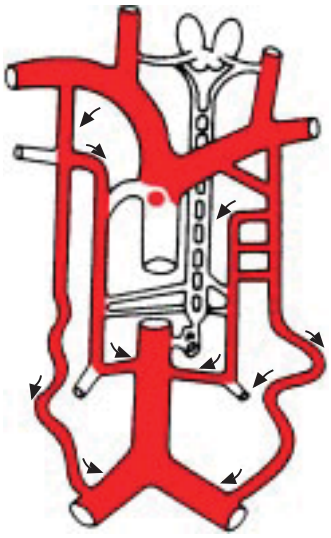
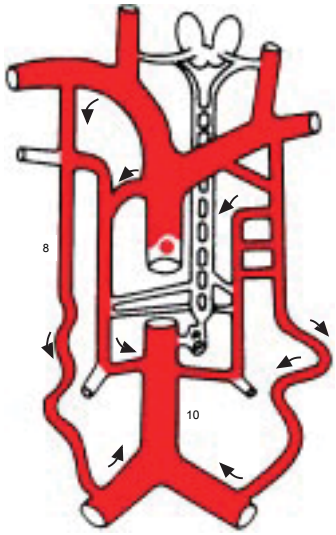


Fig. 38.2 Obstruction of the superior cava at the level of the mouth of the azygos vein: 4, superior vena cava:
5, azygos minor vein: 6, intercostal veins; 7, internal mammary vein.

Fig. 38.3 Obstruction of the superior cava below the mouth of the azygos vein: 8, thoracoepigastric vein;
9, abdominal subcutaneous vein; 10, inferior vena cava.



deep and not visible. The blood that descends through the superior cava is poured through the azygos vein towards the ascending lumbar vein, passes to the iliac veins and from there to the inferior vena cava (fig. 38.3).

Arterial collateral circulation is interesting, especially in a fairly well-known disease that has been called aortic coarctation or narrowing of the aortic isthmus that gives rise to arterial circulatory difficulty in which collaterals develop to try to make up for this defect. Thus the mammary glands become visible,

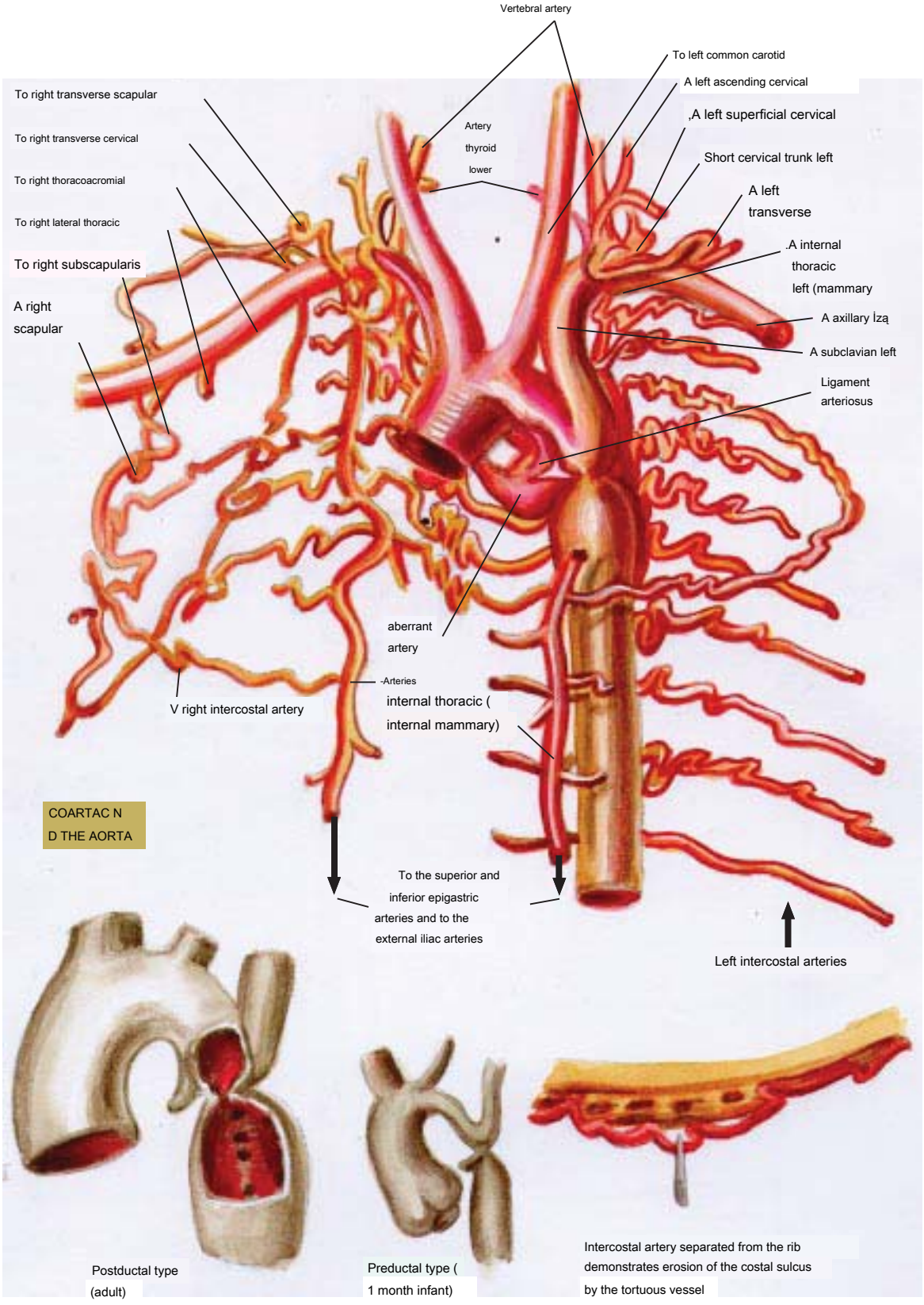

Vertebral artery
To left common carotid
A left ascending cervical
,A left superficial cervical
Short cervical trunk left
A left transverse
.A internal thoracic left (mammary
A axillary İzą
A subclavian left
Ligament arteriosus
To right transverse scapular
To right transverse cervical
To right thoracoacromial
To right lateral thoracic
To right subscapularis
A right scapular
Artery thyroid lower
aberrant artery
-Arteries internal thoracic (internal mammary)
V right intercostal artery
COARTAC N D THE AORTA
To the superior and inferior epigastric arteries and to the external iliac arteries
Left intercostal arteries
Postductal type (adult)
Preductal type (1 month infant)
Intercostal artery separated from the rib demonstrates erosion of the costal sulcus by the tortuous vessel

Fig. 38.4 Coarctation of the aorta: arterial collateral circulation, types and Roessle's sign (notch on the anterior surface of the ribs due to arterial collateral circulation).

although sometimes you can see the whole glass. We have already said that this arterial collateral circulation sometimes affects the ribs, where it determines notches that, using radiography, lead us to the diagnosis (Roessle sign) (fig. 38.4).

EDEMA

It has already been studied in this book, in the “Generalities” part.

CLUB FINGERS OR IPOCRTIC FINGERS

The general examination of the patient provides data on other aspects of great interest, thus the examination of the extremities allows us to detect a special deformity of the last phalanges, which become more or less regularly spherical and accompanying this process, the nails become convex, like watch glass, with a more or less visible cyanosis at that level, which is known as name for clubbing fingers or hippocratic fingers. They almost always accompany cyanotic congenital heart diseases and are also seen in certain chronic respiratory processes. These alterations seem to be due to trophic disorders resulting from anoxemia. They have already been studied in the respiratory system.

CONSTITUTION

Another interesting fact is provided by the constitution. It will be easy to understand that the heart of a long-line will not be the same as that of a brevi-line; It is enough to remember that in the normal individual, the heart follows an oblique direction from back to front, from right to left, from top to bottom, forming approximately an angle of 45°.
In the longilinear subject it tends to be vertical. The cardiac development itself is less, assuming an aspect that causes it to be called a gouty heart. On the other hand, in brevilines the major axis tends to become horizontal. Almost always in these individuals the heart is also larger in volume.

CIRCULATORY SYSTEM. ALTERATIONS IN THE PRECORDIAL REGION

39

As we continue the study of circulatory semiology, we will consider the alterations in the exploration or examination of the heart and the great vessels in the precordial region.

Before beginning this chapter, and the following ones related to the physical examination of the precordial region, it is advisable that you review the circulatory anatomy and physiology and the examination of the precordium, discussed in Chapters 10 and 11, respectively, of Section I.

INSPECTION

The inspection and palpation technique is simple and has already been explained previously.

Through the inspection we will observe all the visible normal and abnormal phenomena, such as the coloration of the skin, the architecture of the region, its external configuration, and the heartbeats, whether positive, in the sense of rising, or negative, in the sense of depression, both in the precordial region and in the epigastric and cervical.

Static and dynamic inspection are carried out in the precordial region.

ESTICAL INSPECTION

Semiography and semidiagnosis

Due to it, some pathological deformations of the thorax linked to certain cardiovascular conditions can be discovered, among which we have kyphoscoliosis, lordosis, pectus excavatun, etc., which when accentuated can cause heart disease, due to the disorders that they usually cause in the minor circulation and in the functioning of the organ itself.

In children, in whom the normal ossification process of the ribs has not yet finished, marked growth of the heart or abundant pericardial effusion usually bulges the precordial region and deforms the thorax.

In adults, aneurysms of the ascending aorta, of the arch of the thoracic aorta, can externalize by corroding the tissues that surround them, including the bone structures (ribs and sternum) as a rounded mass that pulsates and expands synchronously with cardiac systole.

As soft tissue anomalies we will mention gynecomastia in patients taking digitalis, almost always in elderly subjects with a relative deficit of androgenic hormones; and polythelia (supernumerary nipples), which is frequently observed in some congenital heart diseases; Finally, we can also observe the collateral venous circulation, laterothoracic, in thrombosis of the superior vena cava, and the dilation of the superficial arteries in artic coarctation.

DIN MICA INSPECTION

Cou ue of the tip

Semiography and semidiagnosis

It allows us to appreciate if there are alterations in the tip shock in relation to its frequency, rhythm, location and shape. Frequency and rhythm will be assessed later in the study of arrhythmias.

In certain conditions, the impingement of the tip moves upward in intra-abdominal hypertension, due to pathological processes contained in its cavity (ascites, marked hepatomegaly, giant ovarian cysts, etc.) and in pericardial effusion it can even reach the third space; It moves downward in hypertrophy of the left ventricle (to the sixth or seventh space) and when the heart is rejected by an aortic process (aneurysm of the posterior thoracic aorta).

The tip shock moves to the left in hypertrophy and dilation of the right atrium or ventricle, in effusion or pneumothorax of the right pleural cavity, and in fibrous or atelectatic retraction of the left lung. The shift to the right is produced by the presence of abundant fluid or air within the left pleural cavity and by pulmonary fibrosis or atelectatic retraction of the right lung.

In global heart failure the shock is perceived downwards (sixth or seventh space) and outside the midclavicular line.

In patients with dextrocardia, apex impingement is observed in the right hemithorax.

Form, intensity and extension

Remember that the intensity of the tip shock depends on the thickness of the wall, the size of the heart and the strength of its contraction.

Usually the increase in intensity of the tip shock corresponds with a greater extension of the shock, which can occur, independently of cardiac growth, when there is erethism of the heart (after exertion, in neurotics and hyperthyroid patients).

The form and character of this shock can be pathologically altered, causing the following modalities:

-Negative heartbeat.

-Ladder beat.

-Dome heartbeat.

- Universal heartbeat.

In the negative beat, the shock of the apex, far from lifting the precordial region, paradoxically depresses it, in each ventricular systole.

This is seen in right ventricular hypertrophy.

In this case the right ventricle will be part of the apex of the heart. When ventricular systole occurs, the right ventricle, which is forming part of the tip of the heart, moves away from the chest wall and twists on its longitudinal axis from left to right and from front to back. A vacuum is produced that sucks in the chest wall, giving this systolic depression of the tip which is known as a negative heartbeat.

In the ladder beat you see two weak beats at the top instead of one. The cause of this phenomenon is the loss of cardiac "tone." Under these conditions, the hypotonic ventricle offers no resistance to the surge of blood falling from the atrium. If the ventricle is very dilated, in the period called rapid filling, that is, at the beginning of diastole, the moment in which the blood that was accumulating in the atrium suddenly enters the ventricle, when the corresponding valve opens, the aforementioned blood surge lifts the wall of the hypotonic ventricle, giving rise to the weak second beat that characterizes the so-called ladder beat, and which in this case will be protodiastolic.

The dome beat is an intense, vigorous shock that gives the impression upon palpation that we are in the presence of a billiard ball or dome, and which has been described with the name "Bard's dome shock." It appears in large hypertrophies of the left ventricle, characteristics of aortic insufficiency because this is the valve disease that causes greater hypertrophies of said ventricle.

The universal beat is a global phenomenon in which the entire precordial wall is projected forward because there is great ventricular hypertrophy; Under these conditions, the organ contacts a large part of its surface with the anterior wall of the thorax; When systole occurs and the anteroposterior diameter of the heart increases, at the same time as this organ rotates from left to right and from front to back, a diagonal beat is created that runs obliquely through the precordial region and is called the universal heartbeat.

In the absence of cardiac hypertrophy, this phenomenon can be seen when the heart is wedged between the precordial region and a retrocardiac or prevertebral tumor that, pushing this normal-sized heart forward, will cause the same phenomenon when ventricular systole occurs.

Other beats unrelated to the cu ue of the tip

In pathological conditions, the epigastric beat can be observed due to the transmission of the aortic beat; It is understood that patients with high differential pressure, as a result of aortic insufficiency, persistence of the ductus arteriosus, or marked anomalies, etc., whose aortic beat is wide, commonly present an epigastric beat, which is the transmission of the exaggerated aortic beat. Hyperthyroidism, neurocirculatory asthenia, among others, in addition to cardiac erethism due to physical effort and emotions in healthy subjects, favor its appearance.

The negative epigastric beat (systolic retraction of that region) occurs in the pathological field, when there is hypertrophy of the right ventricle.

Finally, less frequently, the epigastric beat is linked to the existence of a true hepatic pulse, of ventricular origin, when there is tricuspid insufficiency.

In abnormal conditions, a heartbeat can also be observed in the left second intercostal space (site where the pulmonary artery projects when its trunk dilates), which occurs in the course of diseases that generate pulmonary arterial hypertension of sufficient magnitude to dilate this vessel and allow its intimate contact with the chest wall, which gives rise to a visible heartbeat in the left second space. This occurs in acute and subacute cor pulmonale, in rheumatic heart disease with damage to the mitral valve, etc.

Independently of the impingement of the tip, as a result of an aneurysm of the ascending aorta, a heartbeat can also be observed in the second or third right intercostal space, before the ectasia perforates the chest wall and causes the appearance of a "tumor."

Finally, visible beats of the intercostal, internal mammary or scapular arteries can be identified; These arteries can develop abnormally by a compensatory mechanism when there is coarctation of the aorta.

PALPATION

Palpation of the precordial region completes the inspection and provides new data, some of it of considerable diagnostic significance.

SENSITIVITY OF THE PRECORDIAL REGION

Before thinking about referred pain of cardiac origin, it is necessary to rule out all conditions of the musculocutaneous covering: panniculitis, myalgia, osteitis, arthritis, neuritis, etc.

Digital pressure awakens pain at the level of the phrenic points in acute pericarditis; in the second left intercostal space and at the level of the manubrium of the sternum, in aortitis, and in the projection area of the atrium, in the posterior wall (interscapulovertebral space at the level of D2-D4) in mitral stenosis (posterior auricular point of Vaquez).

SEMIOGRAPHY AND SEMIODIAGNOSTICS

Below we will describe the most important pathological elements obtained during palpation:

- Alterations of the tip shock.
- Palpable valve vibrations (valvular shocks).
- Catharic thrill.
- Diastolic gallop rhythm.
- Palpable pericardial rubs (pericardial fremitus).

Alterations of the cu or ue of the tip

In certain cases of hypertrophy and dilation of both ventricles, the precordial region is projected, as a whole, forward, with great energy, during cardiac systole, constituting the universal shock, already described in the inspection as a universal beat; Other times, in left ventricular hypertrophies, mainly those caused by aortic stenosis, a sui generis apex beat is identified by palpation, the Bard's dome shock, which consists of a beat that is slightly wider than normal and gives the sensation as if a small balloon was inflating and deflating following the rhythm of the heart.

Other times, the rise or fall of the tip shock is not done in two stages, as a result of the addition of a jump given by a low-energy palpable vibration, presystolic or protodiastolic, which constitutes the palpable gallop. When we refer to the auscultatory method, we will make its detailed clinical description and semiological assessment.

Palpable valve vibrations with valvular ues

When palpating the tip, a dry, brief and distinct shock can be perceived, which, because it occurs during systole, is called systolic valve shock; It is usually found in mitral stenosis; It is the tactile manifestation of the brilliance of the first sound and, as it results from the closure of a sclerosed mitral valve apparatus, it constitutes the closing click of the mitral valve. In eretic hearts (young, hyperthyroid and sympathicotonic), a similar, but less marked, systolic vibration can be seen. Well-marked systolic valve vibration, outside of cardiac erethism, is attributed almost exclusively to old rheumatic valvulitis with mitral stenosis. When shock is present

systolic valve of mitral stenosis, it is advisable to look for its temporal relationship with apexal uplift. In intense stenosis it is usually delayed, in relation to the beginning of systole marked by the heartbeat.

If we palpate the same region of the tip, another valve vibration can be perceived, while the tip shock decreases, a little after diastole has begun. It is also a brief and dry shock, but because it occurs in protodiastole it is called protodiastolic valve shock; This valve click corresponds in time to the opening of the mitral valve and constitutes the tactile manifestation of the mitral opening click, observed in old mitral valvulitis, with stenosis of this atrioventricular orifice.

By palpating the base, the heartbeats we refer to in the inspection can be identified; In addition, the existence of expansion can be verified when there is an aneurysmal tumor, or palpable valve vibrations arising in the aortic or pulmonary sigmoid. The expansion of an aneurysmal sac is a clinical sign of great value for allowing the differential diagnosis between a tumor adjacent to a vessel that communicates its heartbeat and an aneurysmal tumor, with its own heartbeat, and, consequently, with expansion. In the first case, the movement is in one direction only; in the second, that of expansion, the heartbeat movement is in all directions, which can be easily appreciated by holding the "tumor" with two or more fingers, in one direction. or both hands (according to the size of the aneurysm), and noticing that the fingers separate with each heartbeat, whichever direction is chosen.

Basal valve vibrations are always diastolic (diastolic valve shocks) and give, to the palpating subject, the sensation of a brief, dry knock at the moment of sigmoid closure. These vibrations reflect hypertension, either of the pulmonary artery (pulmonary diastolic click) or of the aorta (aortic diastolic click), or sclerosis of the aortic valve apparatus (aortic atherosclerosis, aortic syphilis) or pulmonary (pulmonary atherosclerosis due to chronic pulmonary hypertension, as in old mitral stenosis). The causes of arterial hypertension, general or pulmonary, have already been indicated previously.

In the left xiphoid and paraxiphoid region in the fifth, fourth and third intercostal spaces next to the sternum, and in the sternum itself, in its lowest portion, a systolic beat of greater or lesser energy can be identified, in cases of growth of the right ventricle, particularly its ejection chamber.

In that same area, corresponding to the outlet chamber of the right ventricle itself, it has been possible to perceive a systolic valve shock produced by the closure of the tricuspid valve in patients with high hypertension.

right ventricular, particularly in the initial stages of acute cor pulmonale.

These patients, as a result of pulmonary embolism of sufficient magnitude and, perhaps, the reflex constriction of other unobstructed pulmonary vessels (pulmopulmonary reflex), develop marked pulmonary hypertension that causes the right intraventricular pressure and the dimensions of the ventricle itself to grow suddenly, first in its ejection chamber and then in its inlet chamber.

As long as the dilation of the inlet chamber is not so considerable as to sufficiently widen the atrioventricular orifice and cause a so-called functional tricuspid insufficiency (muscular organ), the closure of the tricuspid is done with unusual energy and can produce this palpable systolic vibration, which is always accompanied by another systolic vibration - perceptible in that same area and in the second left space-, of pulmonary closure. sigmoid, secondary to pulmonary hypertension.

These physical signs have often helped to establish the early diagnosis of acute cor pulmonale, before the electrocardiographic trace is seen and the subjective manifestations (cough with hemoptoic expectoration and flank pain) and objective manifestations (physical and radiological examination) of the pulmonary infarction consecutive to the pulmonary embolism are exhibited, in the presence of a picture of circulatory collapse (pulmoperipheral reflex) that is usually accompanied of constrictive precordial pain due to acute coronary insufficiency (pulmocoronary reflex), which contributes to misunderstanding the diagnosis with that of coronary occlusion. The presence of a previous heart disease that causes the right ventricle to grow as a result of an increase in pulmonary blood pressure greatly restricts the clinical value of these palpatory signs for the diagnosis of acute pulmonary heart disease, as occurs in mitral valve patients.

Shudder cathar

The concept, senmiogenesis or pathophysiology, semiotechnics and basic semiography of thrill were already described in Section I. Here we will return to semiography to explain semidiagnosis.

Semiography and semidiagnosis

The base systolic thrill can be located on the right or left of the sternum. In the first case, it almost always corresponds to aortic stenosis, rarely depending on the existence of an aneurysm. In both cases it spreads to the carotids, where it continues to be perceived. Systolic thrill is usually perceived in the carotids,

without it existing in the second right space; This is what commonly occurs in cases of aortic insufficiency, without concomitant real stenosis, or in the presence of syphilitic aortitis with suprasigmoid dilation.

The basal systolic thrill located to the left of the sternum may be due to congenital pulmonary stenosis; to persistence of the ductus arteriosus (atypical), which on auscultation is manifested only by a systolic murmur and not by the characteristic continuous murmur; a dilation of the pulmonary artery with relative sigmoid stenosis, as occurs in atrial septal defect or in accentuated pulmonary atherosclerosis; or it may be due to aortic stenosis.

Mesocardiac systolic thrill usually reflects the existence of a ventricular septal defect, almost always of a congenital nature (Roger's disease). Xiphoid thrill due to organic tricuspid insufficiency is an exceptional occurrence, despite the relatively high frequency of tricuspid valvulitis. Its location is rather in the fourth left intercostal space next to the sternum.

The diastolic thrill most commonly found in clinic is the one located in the apex. It can occupy the entire diastole or only the presystole; It is almost always accompanied by the systolic valve click, which completes the thrill and the protodiastolic click (mitral valve closing and opening shocks).

Apex diastolic thrill is almost pathognomonic of mitral stenosis. In exceptional conditions, a basal diastolic thrill of aortic insufficiency can be transmitted to the apex. In reality, aortic insufficiency rarely results in cathartic shuddering.

In relative tricuspid stenosis due to considerable dilation of the right atrium and ventricle, which converts the right atrioventricular orifice into a relatively narrow ring (organomuscular tricuspid stenosis), we perceive diastolic thrill in the left paraxiphoid area, identical to that observed in organovalvular tricuspid stenosis.

The continuous systodiastolic thrill is located in the second left intercostal space and is almost pathognomonic of persistence of the ductus arteriosus.
Sometimes, the thrill is more intense during systole, but in others, the reinforcement is diastolic.

In summary, when considering all the general causes of continuous thrill production, four main ones are listed: the persistence of the ductus arteriosus, arteriovenous aneurysm, toxic goiter and malignant vascular tumors. But, not from the general point of view, but rather focusing the semiological problem towards the assessment of the continuous thrill, precisely located in the first intercostal spaces, of these four factors mentioned, almost only the first fits, that is, the persistence of the ductus arteriosus. There are other exceptional congenital anomalies that can produce the same palpatory and auscultatory phenomena, the study of which corresponds to specialists.

Nowadays, formulating the diagnosis of this persistence can immediately mean a surgical indication; Hence, the semiological analysis of the signs by which it is identified has enormous interest, greater than the purely academic one.

Diastolic gallop rhythm

It has a tactile expression derived from the ventricular filling wave in presystole or protodiastole. The palpating hand notices a soft, undulating, intense ridge in the region of the tip in the left gallop or in the vicinity of the tricuspid focus, in the right.
In the first case, it can be confused with the brief, dry protodiastolic shock, which occurs at the moment of opening of the stenosed mitral valve; It differs, however, because the vibration given by the gallop is less vibrant, less dry and longer, although the time in which both appear, within the cardiac revolution, is almost the same.

Palpable pericardial friction friction due to pericardial myth

The surface of the pericardial leaves usually thickens as a result of a primitive inflammatory process (frequently rheumatic), toxic (uremic pericarditis) or degenerative (myocardial infarctions that involve the epicardium). In such circumstances, the friction of the thickened and "dull" pericardial sheets gives rise to vibrations that can be perceived by palpation, which are called palpable pericardial friction or pericardial fremitus. This phenomenon is usually heard more frequently at the level of the third and fourth left intercostal spaces, next to the sternum, and its identification is easier through auscultation.

It consists of a sensation of rubbing, both systolic and diastolic, in swinging motion, following the rhythm of the heartbeat. Pericardial rubs are better perceived when the palm of the hand palpating the precordial region is applied strongly to the chest wall than when the pressure is light.

They are common in dry pericarditis and in pericarditis with effusion, before the effusion appears or after its resorption.

COMBINED INSPECTION AND PALPATION IN THE EPIG STRICA REGION

In the epigastric region we collect above all a beat that we call the epigastric beat, of place, characters and

variable origin. First of all, it is necessary to distinguish between two types of epigastric beats; one that is transmitted and reaches the epigastric region spread from the aorta through other organs, or spread from the precordial region, and another type of epigastric beat that belongs properly to that region, because the heart, the aorta or the liver originate it in the epigastrium itself. We call this second type the autochthonous or epigastric heartbeat.

In the transmitted epigastric beat of aortic origin, we observe the systolic rise that characterizes the beat in the center of the epigastrium, more or less at an equal distance from the xiphoid appendix and the umbilicus, and it has another characteristic and that is that this beat is seen, but generally not palpated. The fact that the heartbeat can be seen and not palpated is due to the fact that as soon as palpation exerts a certain pressure, the transmission stops taking place. In the case of epigastric beats transmitted as a consequence of large precordial beats, we observe how the epigastric beat coincides with and is a consequence of the precordial beat. This type of universal precordial beat will be seen above all in large ventricular hypertrophies.

The most interesting epigastric beats are the autochthonous ones, those that originate there in the epigastric region.
First, we have the indigenous epigastric beat of aortic origin. It is perceived in a subject who has a lesion of the aorta and this comes into direct contact with the epigastrium. There is sclerosis of the abdominal aorta with decreased elasticity of the vessel; This is sinuous. The heartbeat becomes more visible when the aorta is dilated, or when there is an abdominal aortic aneurysm. In these cases it will be very easy to understand that there will be a systolic arterial epigastric beat, positive, which obviously corresponds to an alteration of the aorta: autochthonous epigastric beat of aortic origin.

It is also palpable, and palpation gives, together with the increased sensitivity of the vessel, the beat that allows the process to be recognized when the abdominal wall does not make it visible; In addition, you can take the aorta with your hand, notice its volume, feel the expansion of the vessel, which should serve as a differential element, and to a certain extent, notice its flexuosities and the length of its abdominal path.

The autochthonous epigastric beat may be of cardiac origin. It is the right ventricle that determines it.
When the right ventricle hypertrophies, or hypertrophies and dilates at the same time, it is understood that its epigastric relationship is closer, and, naturally, in these cases, with a normal wall, there is nothing strange in verifying that the right ventricular systole is manifested by an epigastric beat that occupies the highest part of the epigastrium, that is, the place that remains delimited.

midway by the convergence of the costal arches, immediately below the xiphoid process somewhat to the left. Finally, it will have a character that serves to avoid confusion with the aortic beat and that is that, in addition to the site where it occurs, the right ventricular beat is negative; It is a systolic depression that is explained by the same reasons that served to explain the negative precordial beats.

There is a third type of autochthonous heartbeat of great interest, it is the one determined by the hepatic pulse.
The relationships of the liver with the right cavities are very close, especially with the right atrium. We must highlight as clearly as possible the close relationship that exists between the liver and the right atrium and therefore, the right heart.
The liver is a large blood lake, which is reached by the portal vein, forming a portal system, which ends in the suprahepatic veins, which drain into the inferior cava, almost emptying into the right atrium. This means that as soon as there is difficulty emptying the right atrium, return circulation is affected and the liver increases in size. This process will appear in all those cases in which the right ventricle is disturbed in its work: when there is tricuspid insufficiency and, more frequently, when pulmonary hypertension occurs and the right ventricle suffers.
The hepatic pulse becomes more evident when, as a consequence of one of these processes, the right ventricle dilates and the tricuspid is no longer able to close an atrioventricular orifice that is abnormally large, even when it is normal; This gives rise to tricuspid insufficiency that we call functional because it does not respond to any injury. It then happens that during ventricular systole, blood is injected into the right atrium, which hinders return circulation and, sometimes, a systolic hepatic pulse with systolic beats, which may be palpable, is perceived in the liver.

COMBINED INSPECTION AND PALPATION IN THE CERVICAL REGION

Finally, we have the study of inspection and palpation of the cervical region, in which we also collect data of interest. There are four main elements that we will study:

-Venous beats.

-Arterial beats.

- Cardarelli-Oliver sign.

- Venous repletion.

VENOUS BEATS

They can be observed in normal conditions on the neck. Especially in the jugular fossa, it is possible to see a type of venous beat that coincides with presystole and is negative: physiological or auricular venous beat. In pathological conditions the venous beats become much more visible and important and are perceived in other regions of the neck; This beat is positive, and is called a pathological ventricular venous beat.

In functional tricuspid insufficiency, the venous pulse becomes systolic, which is why it will be more visible, since, during the moment of ventricular systole, the venous vessel is distended due to an abnormal injection of blood into the atrium, as a consequence of the incapacity of the tricuspid valve. As functional tricuspid insufficiency is difficult to recognize by auscultation or other procedures, both the systolic venous pulse and the hepatic pulse are of the highest clinical diagnostic value in this entity.

ARTERIAL BEATS

They can be seen on the neck. They are generally systolic beats that are due to the carotid. It is possible to find them in very diverse pathological conditions.

Firstly, in vascular erethism produced by emotional states or sustained physical effort, or in cases where there is a disturbance of the vegetative nervous balance. Thyroid hyperfunction is the one that most frequently causes this symptom. Secondly, the lesions of the vessel, arterial sclerosis, mainly of the carotid. But the arterial beats will be visible above all in the neck, when there is a certain valvular lesion, especially suitable for producing these high amplitude beats: aortic insufficiency. In aortic insufficiency, blood will reflux during diastole from the aorta to the left ventricle, which results, together with left ventricular hypertrophy, in the separation of the two blood pressures being very large, that is, there is a large differential pressure. This naturally means that the vessels, in general, are showing a marked process of distension and depletion that makes them beat in a violent way. As this process is not limited to the carotid arteries, but is visible in other arteries that under special circumstances can beat in the neck, such as the subclavian arteries, the lower thyroid, and the aorta itself above the fork of the sternum, such abundant and broad beats are seen in these cases that they have been called arterial dance.

We have already said that it is possible to observe other times that it is the aorta that beats above the fork in the neck. The one that interests us most is the beat that we are going to perceive above the sternum fork in cases where there are alterations of the aortic wall: aortic sclerosis, aortic atheromas and syphilitic aortitis.

As a consequence, especially of sclerosis, the aorta loses its elasticity, increases in length and the arch protrudes behind the fork of the sternum. Then we see it beating. When the heartbeat is visible and, furthermore, we feel it when palpating above the fork, we are sure that the aorta itself determines it directly for any of the reasons indicated. It is necessary to be aware that on some occasions when the suprasternal beat is not seen with all the characteristics of the aortic beat, it is a type of transmitted beat, which is not palpable and in reality there are no alterations of the vessel that determine it. Sometimes above the clavicles we may see the beating of the subclavian arteries: these are almost always processes of aortic sclerosis or sclerosis of those same vessels, which in a certain way makes them more visible. Aneurysms, when located at this level, will be a cause of visible and palpable arterial pulsation.

CARDARELLI OLIVER SIGN

This sign was described independently by Cardarelli in 1872 and by Oliver in 1878. It consists of traction movements of the trachea in each systole.

The technique to observe it described by Osler is as follows: the patient is placed in an upright position, ordered to close his mouth and raise his chin as much as possible. The cricoid cartilage is then grasped between the index finger and the thumb and gentle upward pressure is exerted on it. When there is dilation or aneurysm, the pulsation of the aorta transmitted to the hand through the trachea will be clearly perceived.

It constitutes a very important sign for the diagnosis of aneurysms of the transverse portion of the aortic arch.

VENOUS REPLETION

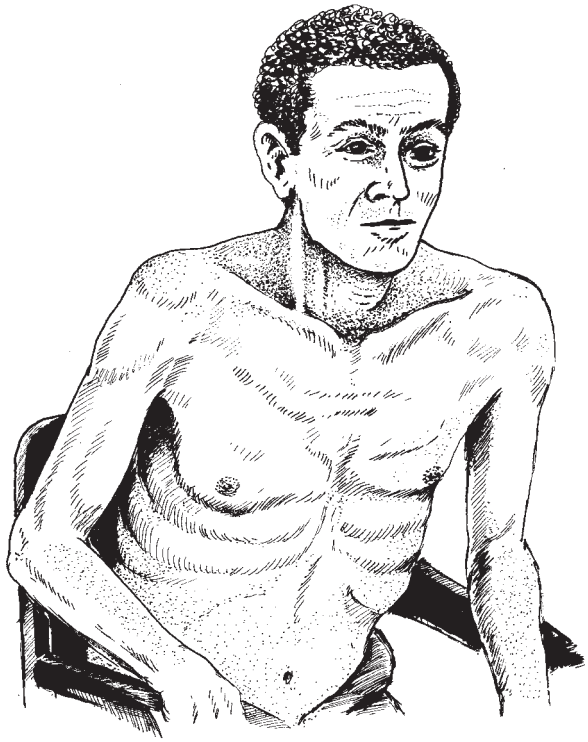
The most important cervical veins are the external jugular and internal jugular veins. These veins are very close to the mouth of the right atrium. They reflect, in a very constant way, the pressure changes of the right atrium. If we lie down a normal subject, the trunks of his veins, both the internal jugular veins and the generally more visible external ones, will become distended, they will be marked under the skin, as a consequence of venous engorgement. It is a physiological fact, because in reality the veins and the right atrium are communicating vessels. In this horizontal position, the pressure in the veins is greater because they are in a situation that is not favorable for drainage into the corresponding atrium:

are below the phlebostatic level.

On the contrary, if we make the subject sit up using pillows, until we end up sitting him down, we see how during this progressive ascent towards the vertical position, the veins in the neck become erased until they end up disappearing. In such a way that in the sitting position no vein is visible in the cervical region. But if pathological alterations of intra-atrial venous pressure occur, then, when we lift the subject, the venous engorgement in the neck will not disappear, and even sitting, we will still see two small raised venous trunks, one on each side of the neck, which accurately demonstrate the existence of hypertension within the atrium (fig. 39.1).

Generally, at the free end of this blood column, we will perceive the venous beats, which will be a valuable element of identification. When we find engorged cervical veins with the individual in a position close to the vertical, and they are animated by beating, we have a sign of extraordinary value to ensure that there is venous hypertension, that is, that there is a stasis, a difficulty in emptying the right atrium and, therefore, in the drainage of the peripheral venous circulation. This simple maneuver of great value almost allows the measurement of intra-atrial pressure, if not with extraordinary precision, at least approximately.

Fig. 39.1 Jugular engorgement.



PERCUSSION

The general considerations and technique of percussion of the precordial area were already discussed in Section I.

Increase in the area of relative dullness Semiodiagnosis

When the left ventricle is enlarged, the area of dullness extends outward and downward, and the upper limit of the left border usually reaches the third intercostal space. If the right ventricle is dilated, the limits of the cardiac silhouette also move outwards and downwards. The dilation of the right atrium causes the lower edge to be percussed outside the line that passes through the edge of the sternum in the fourth, fifth and sixth intercostal spaces.

The aneurysm or dilation of the ascending portion of the aorta determines the dullness of the manubrium of the sternum and the widening of the vascular pedicle, which is percussed outside its normal limits.

In large dilations and aneurysms of the pulmonary artery there is an area of parasternal dullness in the left second and third spaces.

Increase in the area of absolute dullness Semiodiagnosis

When the right ventricular outflow tract is enlarged (Fig. 39. 2), the area of dullness extends upward to the third intercostal space immediately lateral to the edge of the sternum. If the entry tract is dilated, it produces dullness of the sternal body that can extend to the right in the fourth, fifth and sixth intercostal spaces. The notable enlargements of the right atrium (Fig. 39.3) determine that the area extends outside the right border of the sternum in the fourth, fifth and sixth spaces.

In aneurysms or large dilations of the ascending aorta, of the pulmonary trunk, or in tumors of the anterior mediastinum (goiter, thymomas, dermoid cysts, etc.) the manubrium of the sternum, which is normally sonorous, may become dull in a more or less extensive area.

Pericardial effusions notably increase both areas (Fig. 39.4), may even cause areas of dullness in the posterior region of the chest, and can be confused with pleural effusions or other lung lesions.

Marked emphysema can mask both areas of dullness.

AUSCULTATION

The generalities of precordial auscultation have already been described in Section I. In this chapter we will do

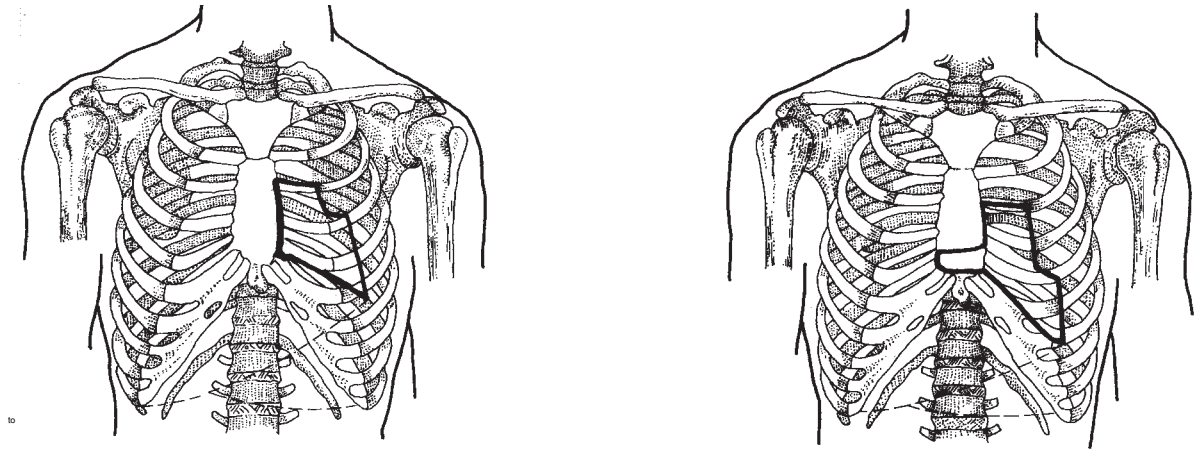


Fig. 39.2 Increased area of absolute dullness: a, when the right ventricular outflow tract is enlarged; b, When the inlet and outlet tract is enlarged.

Fig. 39.3 Enlargement of the right atrium.

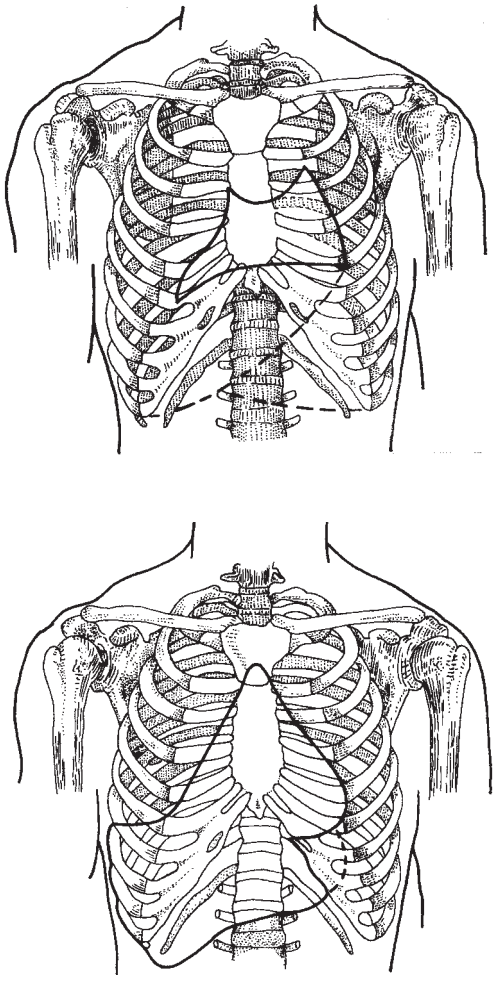


Fig. 39.4 Pericardial effusion.

some considerations that were not made previously and we will reiterate others that are important and that serve as a basis for the study of auscultation of alterations.

of heart sounds and murmurs, which will be described in the next two chapters, respectively.

USE OF THEESTETHOSCOPE

Quiet environment

Many of the defects in auscultation result from the fact that it is performed hastily, in a noisy environment. To carry out a correct auscultation, the room must be quiet. There has often been the experience of hearing a faint murmur one day, and then it is absent the next. A very common cause is the variation in ambient noise. When one person is listening, the rest of the group must remain silent. It is necessary that the patient be comfortable and in a warm environment, to avoid extra muscle noises.

Frequently, the subject must stop breathing.

tion while being auscultated.

Position of the examinee

To perform a complete examination, auscultation must be performed with the subject in three successive positions: sitting, lying down and left lateral decubitus. It may be sufficient to auscultate the subject only in supine position, but the examination will never be conclusive if only the seated person was auscultated. Typical diastolic murmurs, which are clearly heard when the examinee is lying down, may not be heard when the examinee sits up. While lying down, the subject's arms should not be raised above the head as this raises the rib cage and decreases the intensity of heart sounds. The most convenient positions for auscultating the different murmurs will be indicated later.

Although every doctor uses a stethoscope, few know that when using the bell receiver, what they hear varies depending on the pressure with which they apply it to the chest wall. Applying it more gently they hear better

low-pitched sounds, such as heart sounds - especially the first and third -, many gallop rhythms, and the mid-diastolic murmur of mitral stricture.

By increasing the pressure on the receptor, the intensity of these sounds decreases, especially in people with a large fat pad. Mid-diastolic murmurs and the third heart sound suffer variations in intensity to such a degree that they require a certain capacity of the ear to be heard. The third noise is rarely perceived, due, in part, to the fact that the stethoscope is frequently applied with too much pressure. This phenomenon has been explained in the following way (Rappaport and Sprague): when the bell is applied to the skin, it becomes more tense and its natural oscillation period increases. This reinforces the response to higher tones, but at the same time produces a general decrease in the sensitivity of the skin, which in this case acts as the diaphragm of the bell. As a result, the lower frequency components of the heart sounds are attenuated, while the higher frequency components can still be heard well.

This important maneuver, which allows the intensity of low-pitched noises to be reduced by increasing the pressure of the bell on the skin, has certain implications and applications:

1. In order to hear weak, low-pitched noises, the examiner should lightly rest the receiver on the rib cage.

2. By observing how a noise or murmur changes, depending on the greater or lesser pressure exerted on the bell, the pitch of a murmur or noise can be judged to a certain extent.

3. The systolic murmur of mitral regurgitation, which is high-pitched, is not affected as much by pressure changes as medium-pitched physiological murmurs.

4. By increasing the pressure, the masking effect of the first sound on the systolic murmur of mitral insufficiency is reduced, since this, being weak and high-pitched, is not modified by this maneuver as much as the first sound. The same happens with the protodiastolic murmur of aortic insufficiency, which is high in pitch and has a loud second heart sound.

5. In some very "noisy" hearts, with systolic and diastolic murmurs, it is difficult to determine if there is a low-pitched diastolic murmur, due to the intensity of the noise in systole; By increasing the pressure, all sounds are muffled and the diastolic murmur may disappear.

Alternately varying the pressure and concentrating By paying attention to diastole, the examiner can detect the diastolic murmur.

PHONOCARDIOGRAPHS

With the improvement of instruments for recording noises and heart murmurs, and with the greater understanding of physical phenomena, a new field for research work was opened. Several advantages are evident with these recording instruments:

1. Amplification allows a more complete study of weak noises.

2. Permanent recording of the entire frequency spectrum is possible.

3. The simultaneous recording of other cardiac phenomena allows cardiac sounds and murmurs to be safely located.

4. With the use of filters, vibrations of chosen frequencies can be recorded. This record allows the study of the characteristic frequency of each murmur. By amplification of certain frequency bands (selective phonocardiogram) some murmurs may become evident.

Although phonocardiography is an excellent means of study and research, it is not used in daily auscultation. From a clinical point of view, its advantages can be seen in congenital heart diseases.

The value of a phonocardiogram depends on the person who records it. It would be worthless for a simple assistant to place the microphone at a certain point on the chest and take a record.

Records may vary with:

- Small variations in the location of the receiver.
- Amplification.
- Background noises in the device, in the place or in the person being examined.
- Arbitrary modifications to the recording device and the skill with which it is used.
- Type of receiver.
- Structural factors of the examinee.

Spectral phonocardiography is a technique that differs from conventional phonocardiography mainly because it reinforces the frequency spectrum of heart sounds and murmurs, allowing better definition of their timbres.

CIRCULATORY SYSTEM. CHANGES IN HEART SOUNDS

40

The concept, semiography and semiogenesis of heart sounds were already studied in Section I.

CHANGES IN HEART SOUNDS

We will consider the modifications of heart sounds in relation to their intensity, timbre and synchronism.

MODIFICATIONS IN THE INTENSITY OF THE FIRST AND SECOND NOISES SIMULTANEOUSLY

Semiognesis or pathophysiology

1. Extracardiac factors:

a) Thickness of the chest wall. The noises are louder in thin people than in obese people.

b) Position or attitude of the patient. Sounds are perceived more clearly with the person leaning forward, especially in the presence of interposing media: pericardial or pleural effusions, pulmonary emphysema.

c) Acoustic factors. Dependent on the environment, the stethoscope or the observer.

2. Cardiac factors:

a) Intensity of ventricular contraction.

b) Frequency of ventricular contraction.

These two factors have a directly proportional relationship with the intensity of the noises.

Semidiagnosis

1. Increase in the intensity of both noises:

a) Extracardiac factors:

Thinness.

Retractile fibrosis of the pulmonary tongue.

Retrocardiac tumors.

b) Cardiac factors (cardiovascular erethism):

Physical exercise.

Anxiety states.

Hyperthyroidism.

Anemia.

Fever.

2. Decrease in the intensity of both noises:

a) Extracardiac factors:

Obesity.

Edema.

Myxedema.

Left pleural effusion.

Pulmonary emphysema.

b) Cardiac factors:

Constrictive pericarditis or with effusion.

Myocarditis.

Acute myocardial infarction.

States of shock.

Pre-agonic states.

MODIFICATIONS IN THE INTENSITY OF THE FIRST ISOLATED NOISE

Semiognesis or pathophysiology

1. Position of the atrioventricular valves at the time of ventricular contraction, expression of the duration of the PR interval. A short PR determines early closure, with widely separated valves that produce an intense first noise when closed. A long PR determines the opposite phenomena.
2. Structural changes in the atrioventricular valves. Valvular sclerosis produces an intense first noise, while inflammation of the valve causes the noise to dull.

3. Intensity and frequency of ventricular contractions.

Semidiagnosis

1. Increase in the intensity of the first noise:

a) By short PR:

Complete atrioventricular block.

Nodal rhythms.

Atrial flutter.

In Wolff-Parkinson-White syndrome there is no produces alteration at the first noise.

b) Due to mitral stenosis.

c) Due to cardiovascular erethism:

Physical exercise.

Anxiety.

Hyperthyroidism.

Anemia.

Fever.

2. Decrease in the intensity of the first noise:

a) By long PR:

First and second degree atrioventricular blocks.

b) In the period of state of endocarditis and valvulitis.

c) In constrictive pericarditis or with effusion, myocarditis, cardiac infarction, shock, preterm states.

d) Masking by intense systolic murmurs of regurgitation.

MODIFICATIONS IN THE INTENSITY OF THE SECOND ISOLATED NOISE

Semiognesis or pathophysiology

1. Changes in closing pressure in the aorta or pulmonary artery.
2. Structural changes in the sigmoid valves.

Semidiagnosis

1. Increased aortic second sound:

a) Cardiovascular erethism.

b) Severe arterial hypertension.

2. Increased second lung sound:

a) Pulmonary hypertension, in which it can sometimes be palpated (closure of the palpable pulmonary valve) and also dullness can be found on percussion at the level of the auscultation focus, which constitutes the triad known as Chávez complex, of great value for the diagnosis of hypertension in the minor circuit.

3. Decrease in the second aortic sound:

a) Aortic stenosis.

b) Severe arterial hypotension or shock.

4. Decrease in the second lung sound:

a) Pulmonary stenosis.

MODIFICATIONS IN THE NOISE RINGER

Accompanying changes in intensity, changes in the timbre of heart sounds may occur. These modifications are related to changes

structural defects in the valve apparatus and with the presence of gas collections in the natural cavities, which act as resonance chambers.

The alteration in timbre can affect both heart sounds or selectively the first or the second sound. It occurs much more frequently on the left side of the heart, that is, at the level of the mitral and aortic valves.

Semidiagnosis

1. Alterations in the timbre of both noises:

a) Abdominal distention.

b) Pneumothorax.

c) Pneumopericardium.

2. Alterations of the timbre of the first sound:

a) Mitral stenosis. A first intense noise is produced, with a dry, clicking tone, with a pistol shot.

b) Left ventricular hypertrophy. A loud, dull noise, like a roar, may occur.

3. Alterations of the timbre of the second sound:

a) Arterial hypertension. It can produce a second intense aortic sound with a vibrating, clangorous timbre.

b) Pulmonary hypertension. Although less common, hypertension in the minor circuit can also cause changes in the pulmonary valve that cause a second clangorous lung sound.

PATHOLOGICAL MODIFICATIONS OF THE HEART RHYTHM DUE TO ALTERATIONS IN THE RELATIVE DURATION OF NOISES AND SILENCES

TA UICARDIA

In tachycardia the duration of silences is shortened, especially the great silence, and, therefore, the number of beats per minute increases with a frequency greater than 100/min.

BRADYCARDIA

Here there is an increase in the duration of both the noises and the silences, but above all the great silence is lengthened. The frequency is less than 60/min.

FETAL OR EMBRYOC RHYTHM

In this rhythm the two noises and the two silences tend to become equal, resembling the fetal heart when we listen to it through the mother's womb.

Semiognesis or pathophysiology

The semiogenesis of this rhythm is due to three factors:

1. High tachycardia.
2. Arterial hypotension.
3. Alterations of the myocardial fiber.

Semidiagnosis

This rhythm has a very serious prognosis, and appears in:

1. Serious circulatory processes.
2. Major heart failure.
3. Serious general disorders, such as infections with great intoxication of the cardiac fiber.
4. Traumatic surgical shock.

Although this rhythm is evidence of great cardiac failure, it does not necessarily end with death.

DECREASE OF HEART SOUNDS

Concept

As we already explained, the first heart sound is produced fundamentally by the simultaneous closure of the atrioventricular valves and the second sound, by that of the sigmoid valves.

When the closure does not occur synchronously, the single noise is replaced by a double noise, called splitting.

UNDERSTANDING OF THE FIRST NOISE0

Semiography

The splitting of the first sound is best heard at the tip of the heart. The first component is usually less intense. The splitting can be narrow or wide depending on the time between both components, that is, between the closure of the two atrioventricular valves. When it is very broad, that is, when the second component is very separated from the first, then it is not actually a splitting, but rather a systolic ejection noise, commonly called systolic click, of different origin and meaning. Systolic click is frequently found in cases of stenosis, dilation and coarctation of the aorta, and less commonly of the pulmonary artery.

The onomatopoeia of the splitting of the first sound is lorop-dop.

Semiognesis or pathophysiology

The splitting of the first sound is produced by an asynchronism in the closure of the atrial valves.

loventricular, expression of an asynchronism in the contraction of both ventricles. It may represent a normal physiological variant or be an expression of a delay in the activation of one of the ventricles in relation to disorders in the conduction of the electrical impulse (branch block).

Semidiagnosis

The splitting of the first sound does not have much diagnostic value. It is often a normal finding.
It can also appear in right bundle branch block and, less frequently, in left bundle branch block.

SECOND NOISE SPLITTING

semiography

This splitting is best heard at the base of the heart, in the pulmonary focus. It can also be narrow or wide. It is important to specify whether or not the unfolding is modified by breathing. If it is not modified, we can diagnose a fixed splitting of the second sound, which is usually pathological. When the splitting is only perceived during inspiration or increases in degree with it, it is called inspiratory splitting. Finally, the splitting can be perceived only during expiration (disappearing with inspiration) and is called expiratory or paradoxical splitting.

A wide splitting of the second heart sound should not be confused with the mitral opening click, a noise added to the diastolic silence, in cases of mitral stenosis in relation, as its name indicates, to the opening of the valve. This click is best heard at the tip and endoapex, and has a different intensity, tone, and timbre than the second split sound.

The onomatopoeia of the splitting of the second noise is lob-dorop.

Semiognesis or pathophysiology

Inspiratory splitting is due to the increase in venous return to the right chambers of the heart, caused by inspiration. This physiological increase in pulmonary blood flow determines a slight delay in the closure of the pulmonary sigmoid valves with respect to the aortic valves, with the consequent production of a first split sound.

For the same reason, all conditions that delay the contraction of the right ventricle and, consequently, the closure of the pulmonary valve, also give rise to inspiratory splitting, as occurs with disorders in the conduction of the electrical impulse (right bundle branch block).

Fixed splitting may be related to a constant and pathological increase in blood flow lung, as occurs with congenital heart diseases with left-to-right communication, in particular, atrial septal defect. In these cases, the physiological inspiratory increase in venous return no longer has an effect in maximally filled cavities.

Paradoxical splitting reflects a delay in the contraction of the left ventricle and in the closure of the aortic sigmoid ducts, in relation to conduction disorders (left bundle branch block). This splitting disappears during inspiration because at that moment the closure of both valves becomes synchronous, due to the physiological inspiratory delay of the closure of the pulmonary valve.

Semidiagnosis

Inspiratory splitting is a very common normal finding in young adults. It is also associated with right-wing blogging.

Fixed splitting can be a very valuable sign for the diagnosis of atrial septal defect (ASD), especially if the pulmonary component is reinforced and there is a small systolic ejection pulmonary murmur.

The paradoxical splitting allows us to suspect the existence of a left bundle branch block. It also appears in aortic stenosis.

ABNORMAL AND ADDITIONAL HEART SOUNDS

MITRAL VALVE OPENING CASE

Concept and semiog nesis

The opening of a stenosed mitral valve that maintains a certain degree of flexibility can cause a noise, known as the " opening click" and which occurs between 0.08 and 0.10 s after the second sound. Phonocardiograms of normal people may present some small vibrations at this time, but generally do not produce audible noise. In mitral stenosis, the valve resembles a hammock with a central hole; bowing towards the atrium during systole, and at the end of systole, when the pressure in the left ventricle is lower than in the atrium on the same side, it abruptly inverts, bowing towards the ventricle. The higher the pressure in the atrium, the more forceful the valve moves, causing a louder noise.

Other factors similar to those that accentuate the first sound in mitral stenosis also intervene in the production of this accentuated noise.

semiography

The noise is high pitched and dry in tone with a clicking character. It is similar to the second noise and in some

areas may be more intense than this. It is always accompanied by a very clear and usually accentuated first noise. The opening click can be heard throughout the precordial region, but is best heard between the tip or apex and the left edge of the sternum, at the level of the fourth intercostal space, where it is easily recognized by its dry and sharp character. Unless attention is focused on the click, this sound may be masked in the apex region by other sounds associated with mitral stenosis, such as a reinforced first sound and a middiastolic murmur.

In the pulmonary focus, the opening click must be differentiated from a second split sound. This is generally done by recognizing them separately, based on different changes that both noises undergo during breathing.

Semidiagnosis

It occurs in most cases of mitral stenosis; It is one of the most relevant auscultatory signs for diagnosis and usually appears early.

With few exceptions, clicking does not occur when the valves show marked fibrosis and calcification and lose their flexibility. The noise persists even if atrial fibrillation appears, and in most cases, even after mitral commissurotomy, despite the fact that the clinical results obtained from the intervention have been largely satisfactory.

The interval between the beginning of the second heart sound and the opening click of the mitral valve represents the time elapsed between the closure of the sigmoid valves and the moment in which the pressure of the left ventricle falls below the pressure of the atrium on the same side. Consequently, this interval will be shorter the higher the pressure in the left atrium.
Furthermore, when the pressure in the atrium is increased, the production of the first sound is delayed due to the longer time it takes for the ventricle, during the period of isometric contraction, to equalize the atrial pressure. If left atrial pressure drops as a result of mitral commissurotomy, the delay of the first sound (measured by the interval between the beginning of the QRS complex of the electrocardiogram and the beginning of the first sound) can be shortened and the interval between the second sound and the opening click can be increased. These changes can only be verified through phonocardiograms, but they constitute a simple method of evaluating the results obtained with mitral commissurotomy.

Differential diagnosis

The opening click can be differentiated from a splitting of the second noise by considering the following facts:

1. A splitting of the second sound is heard more intensely in the pulmonary focus.
2. By carefully listening to the lung focus, you can pick up both the second split sound and the opening click. During expiration, the splitting of the second noise is minimal or does not occur, and there is a clear interval between the second noise and the opening click. During inspiration, the unfolding is greater and becomes very evident.
3. The opening click is usually heard well in the aortic focus, where breathing does not affect the intervals separating it from the aortic component of the second sound.
4. While the opening click is always accompanied by a clear or reinforced first noise, the same does not happen with the splitting of the second noise.

The mitral opening click can be differentiated from an accentuated or normal third sound on the basis of the following facts:

1. The third noise is more intense in the tip region, but if it is not very pronounced it will not be easily heard throughout the precordial region. Some of the three-beat protodiastolic rhythms and right gallop rhythms may be more evident at the left sternal border, but occur rarely in mitral stenosis.
2. The third noise, normal or reinforced, is separated from the second noise by a wider interval than the opening click.
3. The third sound is lower in pitch and does not have the dry character of the opening click heard in the left fourth intercostal space.
4. Generally, the opening click is associated with a reinforced first sound and other evidence of mitral stenosis.

GALLOP RHYTHM

Concept

It is a three-beat rhythm (exceptionally four beats) because a third pathological noise is added to the two normal heart sounds, reminiscent of the galloping of a horse when it coincides with a high heart rate.

Semiography and classification

The third superadded noise is of low intensity and low pitch. It is best heard at the tip and endoapex, with the bell of the stethoscope.

It is very important to specify whether the added noise occurs in systole or diastole, which allows this

establish the systolic or diastolic character of the gallop, of different significance. As most gallop rhythms are heard well at the tip and poorly or absent at the base, it is a good technique to start auscultating at the base (aortic focus) and using the mapping method to enter at the tip. If the added sound precedes the second sound, the gallop is systolic and if it appears after the second sound, the gallop is diastolic.

Two varieties of diastolic gallop are recognized according to the moment in which the added noise appears in the great silence.

If this noise immediately precedes (0.04-0.08 s) the first sound, at the end of ventricular diastole (therefore, end-diastolic and presystolic), the gallop is called atrial diastolic, since it is synchronous with atrial systole. On the other hand, when the superimposed sound appears in the first part of diastole (0.12-0.20s after the second sound), in the same position as the third physiological sound, it is then a ventricular diastolic gallop. It is frequently associated with alternating pulse and ladder apex beat.

Semiognesis or physiopathology

The systolic gallop occurs in the presence of an accelerated heart rate, when a third noise is added to the small silence. The origin of this noise or systolic click may be extracardiac, of obscure significance and of little importance.

It can also originate in a narrowed or dilated aorta or pulmonary artery due to sudden arterial filling.

Atrial diastolic gallop is frequently due to a delay in atrioventricular conduction (long PR), apparently related to the shock of the blood flow expelled by atrial systole against the ventricle. It disappears if atrial fibrillation develops (there is no atrial systole).

Ventricular diastolic gallop has a disputed production mechanism. Some point out that it originates from the collision of the blood flow against a flaccid ventricular wall in the phase of rapid ventricular filling.
Others claim that it originates from changes in valve dynamics resulting from a pathological increase in residual blood in the cardiac chambers.

Semidiagnosis

systolic gallop

It is frequently benign, innocuous, when its origin is extracardiac. You can also have pathological significance when it appears in the course of stenosis or dilation of the aorta or pulmonary artery.

diastolic gallop

The diagnostic significance is completely different, depending on whether it is an atrial or ventricular diastolic gallop.

The first case is found in the presence of first and second degree atrioventricular block. Also sometimes associated with high blood pressure.

Ventricular diastolic gallop is a sign of great value for the diagnosis of decompensated heart failure, of any etiology. It appears early in cases of myocarditis, primary cardiomyopathies, acute myocardial infarction, hypertensive crises and other advanced cardiopathies. It is usually a herald of acute lung edema, of which it is a part.

SYSTOLE IN ECHO

Concept

Echo systole is a noise caused by the systole of the atria that is perceived during complete atrioventricular blocks; It is heard most clearly in protodiastole, when atrial contraction superimposes the rapid filling phase.

Semiognesis or pathophysiology

When there is complete atrioventricular block, the atria contract due to the excitations of the Keith-Flack node and the ventricles contract by their own or idioventricular rhythm.

Atrial systoles can affect the first or second heart sound, and also the small or large silence. When they affect the great silence and a dull noise is heard added to the heart sounds, it is called echo systole. This noise is preferably heard on the left edge of the sternum between it and the tip of the heart, a place where there is no lung flap and, therefore, sound transmission is facilitated.

Semidiagnosis

Third degree atrioventricular block (complete A-V block).

SEMIOLOGY OF BLOWS

4 1

The generalities of murmurs were discussed in Chapter 111 of Section I, where the general characteristics to be taken into account in the evaluation of a murmur were also stated, which will be detailed in more depth in this chapter.

SEMIOGRAPHY

As previously stated, the study of murmurs includes the evaluation of eight general characteristics: intensity, tone, timbre, the moment of the cardiac revolution in which they occur, their duration, the place where they are heard most intensely, their propagation or irradiation and the modifications that the murmurs undergo under the influence of breathing, muscular effort, changes in position and treatment.

Below we summarize these characters.

ge erale character of the oplo cardiac

- i Intensity
- i Tone
- Ring
- Timing of cardiac revolution in that are produced
- Duration
- 1 Place where they are heard with greater intensity
- i Propagation or irradiation
- Modifications that suffer
  - ì Position changes
  - i Breathing
  - 1 Effort
  - Treatment

INTENSITY

A good system to determine the intensity of murmurs is that of Freeman and Levine, who classify them into six degrees according to their intensity, with the following notes:

Grade 1....Very weak. Poorly audible Grade 2 Weak. Audible only in silence Grade 3 Moderate. Clearly audible Grade 4..... Intense, can be associated with ill

Grade 5.....Very intense; ill palpable.
It can be heard with the stethoscope partially outside the chest Grade 6.... Maximum intensity. palpable ill, you can hear it, even without the stethoscope

Grade 1 murmurs are only heard when listening carefully, in a quiet environment. Those of grade 6 can be heard even when moving the stethoscope away from the costal wall.

The site of greatest intensity of a murmur must be determined as accurately as possible, since this is generally its most important characteristic. The murmurs produced in the different portions of the heart are transmitted to different sites on the chest wall.

The intensity of murmurs, like that of heart sounds, can be modified by extracardiac factors, a fact that must be taken into account when relating the intensity of a murmur to the degree of valve damage.

It has been proven that the intensity of a murmur can be modified by the speed of the blood flow. Murmurs may be less evident at moderate heart rates, which is due to the close relationship between heart rate, minute volume, and current velocity. This possibility must be taken into account when making comparisons between murmurs heard in different conditions.

A murmur is said to have an increscendo or crescent character when, within any cycle, its intensity increases, and when it decreases it is called a decrescendo or decreasing.

TONE

The pitch of the heart murmur also varies considerably from very low to very high tones. The tone of the murmur is a function of two factors: the speed of blood circulation and the anatomical factor that produces it. Not only the speed of blood circulation influences the tone, but also the nature of the obstacle, in such a way that they will be all the more acute the narrower the orifice that generates them, which is perfectly explained, because the greater narrowing makes the vibration that is generated faster, and being faster, a greater number of vibrations are produced per unit of time, which gives the higher-pitched character of a noise. There is, then, the tone in special relation to injuries.

RING

It is another of the elements that are studied in heart murmurs. It allows establishing quite a few differential diagnoses between one type of murmur and another. The bell

It depends on the injury and its nature. When the injury gives rise to a hard and sclerosed valve, the ringing will have a completely different character than that which corresponds to a recent injury, whose valve is covered with layers of fibrin, not yet consolidated or adherent. It will also be understood that the timbre will be completely different when the murmur is generated because in the ventricular cavity a chorda tendineae of one of the papillary muscles, or a valve segment, is free. The timbre may vary if the murmur originates at the time of systole or diastole. Thus, a series of timbres can be distinguished, among which we have: the soft or aspirational timbre, characteristic of the diastolic murmur of aortic insufficiency, which contrasts with the raspy or harsh timbre of the murmur of organic mitral insufficiency, which is compared to that of the vapor jet murmur, which will also be different from the musical timbre and the piante, which accompany the murmur produced by a valvular tear; as is also completely different from that determined, most of the time, by an aberrant tendinous cord or a detached pillar which the bloodstream sets into vibration and give rise to that special timbre, which is called tinbre en guimbarda, similar to the vibration of a metal sheet held between the teeth. They are all completely different timbres.

TIMING OF THE CARDIAC REVOLUTION IN THE EU OCCURRED

It is not necessary to emphasize its importance.
Within this, we must distinguish two types of murmurs:
diastolic and systolic. Systolic murmurs are those that are heard in the small silence, whether or not replacing the first sound, and diastolic murmurs are those that are heard in the great silence, whether or not replacing the second sound.
Systolic murmurs correspond to those lesions in which the passage of the blood column through them occurs during systole.

Other times murmurs will be picked up in diastole.
They will correspond to aortic and pulmonary arterial insufficiencies and atrioventricular valve strictures.

There are times when murmurs called systolic-diastolic are perceived by auscultation of the heart; It is not the association of lesions, which is capable of causing both murmurs to exist at the same time, but rather a special type of lesion that gives a murmur called a continuous murmur, and which corresponds to the persistence of the arterial duct (ductus arteriosus). When this duct persists, a continuous systolic-diastolic murmur is heard in the first or second left intercostal space, 2 or 3 cm from the corresponding edge of the sternum, generally with some reinforcement during systole.

which is characteristic. This murmur has been compared to the noise of machinery and the noise of a tunnel. It is a murmur, a continuous murmur with systolic reinforcement. This is a type of injury that presents a continuous murmur.

DURATION

Duration is another factor that we pay attention to, an essential factor. What do we mean when we talk about the duration of a murmur? We refer to how much it occupies the moment of the cardiac cycle in which it occurs, that is, how much it occupies the systole or diastole, how much it occupies the small or great silence. According to this, we distinguish murmurs into two large groups: one, those that occupy the entire small silence or the entire great silence, and we call those holosystolic or holodiastolic, respectively; The other group includes merosystolic or merodiastolic murmurs, when the murmur occupies a large part of the small silence or the great silence, respectively. These last murmurs are divided into: protosystolic, mid-systolic and end-systolic, and proto-diastolic, mid-diastolic and end-diastolic. End-diastolics are also called presystolics. The duration of the murmurs is an essential character in knowing them.

SITE IN EU ARE HEARD WITH GREATER INTENSITY

One of the characteristics of the heart murmur is the place where its greatest intensity can be heard. This place is usually the precordial region and also the heart auscultation foci that we have studied, but this is not always the case, since the place where the murmur is heard most intensely is the one where it is most easily transmitted, therefore it will vary depending on the place where it occurs. As along with the existence of heart lesions, there are usually alterations in the volume and size of the heart and its cavities, we can explain why the site of greatest intensity does not always correspond to the normal auscultation foci even when the origin of the murmur is valvular, since if it is not valvular it will not be necessary to make a great effort to understand that the place where it is heard with the most intensity should not be a focus of auscultation. It is a very important diagnostic feature. The site is almost always in the precordial region or in neighboring regions.

According to the place where the murmurs are heard with more intensity, we can distinguish the following: tip or apexian murmurs, which are heard with more intensity in the region of the tip; heart base murmurs; murmurs of the xiphoid process or the lower part of the sternum and murmurs of the mesocardiac region or murmurs mesocardiac. In both cases we differentiate them according to the characteristics that we have studied, especially systolic and diastolic.

PROPAGATION OR IRRADIATION

With respect to the propagation of murmurs, the following will be taken into account:

- Factors that determine the location of the site of greatest intensity.
- Factors that influence the propagation of the murmur from the site of greatest intensity.
- Modifications that occur in the timbre of the murmurs during their propagation.

Factors that determine the location of the site of greatest intensity

The site of greatest intensity is determined in principle by the location of the valve involved;
However, the direction of the blood flow that crosses the valve is also of great importance, since, in addition to its vibrations, a large part of the murmur noise depends on the turbulence of the liquid vein. This can be verified by any surgeon by observing on which side of the pathological valve the thrill is perceived. The importance of the direction of the current is demonstrated in aortic valve injuries. In aortic stenosis, part of the murmur originates from vibrations produced in the valves as blood rushes at high speed through the narrowed orifice, while much of it is produced in the aorta by turbulence and shock in the bloodstream. Therefore, the murmur is best heard in the right first and second intercostal spaces. In aortic insufficiency, the murmur is caused by the reflux wave returning to the left ventricle. Most of the murmur is produced at the valve and is best heard along the left edge of the sternum.

Another factor that intervenes in determining the site of greatest intensity of the murmur is the nature of the tissues interposed between the site of origin of the murmur and the chest wall. With the chest open, it can be easily verified that the murmur of mitral regurgitation is heard better over the left atrium; However, this is separated from the chest wall by a tissue that conducts sound poorly, such as the lungs, so the murmur is heard best at the tip, over the left ventricle. In rare cases where the left atrium is dilated and protrudes forward, the murmur may be heard best above the apex, where the atrium contacts the chest wall.

Factors that influence the propagation of the murmur from the site of greatest intensity

The most important factor in the propagation of a murmur is its intensity; The more intense it is, the greater the area in which it can be heard, and some very intense murmurs spread throughout the chest.

The direction of the current that produces the turbulence can determine, to a certain extent, where the murmur is heard. Most of the loud systolic murmur of aortic stenosis occurs in the valve and immediate portion of the aorta; Since it is an intense murmur and occurs near the neck, it spreads well towards it.

Modifications that occur in the timbre of murmurs during their propagation

The reasons why a murmur may change timbre during propagation have been discussed. During propagation, harsh murmurs lose this characteristic and most murmurs acquire a much lower pitch.

EU MODIFICATIONS SUFFER FROM WHISTLES

The modifications that the murmur undergoes are, finally, the eighth of the most essential factors that we consider in the study of murmurs. Due to its importance, we have:

-Position changes.

- Breathing.

- Effort.

- Treatment.

Position changes

Firstly, the modifications that depend on the subject's attitude are considered: a murmur can be heard in the recumbent position and not in the sitting position, or it can be heard in the left recumbent position and not in the others.

They are murmurs that appear or disappear in one recumbent position or another, according to changes in the subject's position.

In general, organic murmurs are constant, they do not change, although there are organic murmurs that are only perceived in the left lateral decubitus position; but, almost always, they do not change or change very little with changes in position; while accidental murmurs, especially anorganic, extracardiac or intracardiac murmurs, are modified by changes in position during the same examination.

Breathing

Murmurs can be modified by breathing. We already said when studying the general mechanism of production, how there were some murmurs that were heard in the region precordial, but which were of cardiopulmonary origin, which depended on the phenomena of aspiration and rejection of air, these changes in the pulmonary tongue being determined by cardiac systole or diastole and by the reduction and increase in the volume of the heart.

It will be well understood that respiratory movements must affect this type of murmur (extracardiac anorganic murmur), in such a way that with a deep inspiration the cardiopulmonary murmurs disappear; On the other hand, lesional organic murmurs are little or not affected by breathing.

Effort

It is another valuable factor in the study of murmurs. There are murmurs that only appear with effort and others that are reinforced by it. In general, organic murmurs become stronger with exertion, because the cardiac systole is stronger and the blood velocity is higher, and we already said that the intensity is a function of the velocity. The effort is also capable of giving rise to inorganic murmurs; These may simply have appeared as a consequence of the increase in blood velocity that exertion determines.

Treatment

We can also point out the modifications that the murmur undergoes under the action of the treatment, and with the variation of the murmur in these circumstances we sometimes obtain the exact interpretation of it. Here it will be observed that almost all lesional organic murmurs increase with treatment. If the subject with the valvular lesion has been in the phase of heart failure, we treat him and as the heart failure improves, as cardiac contractile ability is restored, the murmur increases in intensity, because the systoles become more energetic. On the other hand, the opposite will happen with functional murmurs, which are the consequence of heart failure with dilation. If the dilation is due to heart failure, the treatment by suppressing it will make the valve competent, consequently the murmur will disappear. The diagnosis of some functional murmurs is one of the most important elements. Unfortunately, this is not always the case, because apart from what heart failure adds, there is a permanent increase in the volume of the heart, which is not related to heart failure.

RELATIONSHIP BETWEEN WHISTLE AND FR MYTH

The thrill is the palpable manifestation of a murmur. Since the fingers are much less sensitive and have a lower capacity for discrimination than the ears, the vibrations

tions must be very intense to produce a tactile sensation; Furthermore, by means of this sense the delicate distinction of tone and timbre cannot be carried out.

A thrill doesn't add much to what the ear has already picked up about the underlying injury. The idea that certain lesions, such as aortic stenosis, for example, usually give rise to thrills, has done more harm than good, since many lesions, in which the thrill appears at an advanced stage of evolution, can be diagnosed by correct auscultation long before its palpable expression appears.

Intense and harsh murmurs cause the appearance of thrills more frequently than others, such as the high-pitched murmurs of aortic and mitral insufficiency, which are very rarely accompanied by thrills. Since the fingers perceive vibrations that are below the audible limit (less than 20 cycles per second), low-pitched murmurs should, theoretically, be perceived better by palpation than by auscultation; However, this is very rarely verified in practice.

SYSTOLIC Murmurs

Systolic murmurs are those heard during systole, that is, between the first sound and the second.

Leatham (1958) classified murmurs as follows:

1. Systolic ejection murmurs (Fig. 41.1), which are mid-systolic and are associated with blood flow through the aortic and pulmonary valves. They follow the flow of the normal bloodstream. They include organic, functional and some accidental.

   Aortic stenosis turns out to be the classic ejection murmur referred to by Leatham.

   Pulmonary stenosis is associated with an ejection murmur similar to that of aortic stenosis.

   In addition to valvular or infundibular stenosis of the aorta and pulmonary stenosis, systolic ejection murmurs may be due to these circumstances:

   a) Valve damage without obstruction.

   b) Dilation of the vessel behind the valve.

2. Systolic regurgitant murmurs (see Fig. 41.1), which are holosystolic and produced by mitral or tricuspid regurgitation, or by a ventricular septal defect. They have a direction opposite to that of the blood flow. They can be organic or functional.

   The murmur of mitral regurgitation is the classic regurgitation

   The systolic murmur of tricuspid regurgitation is similar to the murmur of mitral regurgitation.

3. Innocent systolic murmurs, according to Leatham, are due to exaggerated vibrations during the ventricular ejection phase.

4. Diastolic murmurs, seen in mitral and tricuspid stenosis, and in atrioventricular insufficiency. They will be discussed later.

Next, we will describe some of the most common systolic murmurs.

SYSTOLIC TIP MUTURS

Murmur of organic mitral insufficiency

Semiogenesis or

It is caused by organic damage to the mitral valve that prevents its closure during ventricular contraction. Due to this injury, blood passes through the atrioventricular orifice into the atrium.

semiography

Intensity. The intensity is the greatest among all the murmurs heard in the tip region; It can be up to grade 4 or 5.

Tone. The pitch of the murmur is generally high.

Ring. It is variable, from a soft, musical murmur, to the characteristic steam jet murmur, which is the most frequently found.

Moment of cardiac revolution. Systole.

Duration. Holosystolic.

Highest intensity site. According to the location of the lesion, the point or site of greatest intensity is in the focus of the tip.

Propagation or irradiation. If the murmur is of low intensity, it will have a slight propagation in a transverse direction towards the midline; When it is of great intensity it spreads towards the armpit region and can reach the posterior wall of the thorax at the level of the left infrascapular and interscapulovertebral region. It does not spread to the base of the heart.

Modifications. It is modified with changes in the patient's position, listening better in the left lateral decubitus position to increase the contact of the heart with the chest wall in this position. It is modified very little by respiratory movements.

Anorganic or accidental murmurs

They were described in Chapter 11 of Section I.

Murmur of mitral incompetence relative mitral regurgitation

Semiogenesis or pathophysiology

Murmur due to failure of the mitral valve to close the atrioventricular orifice, but without

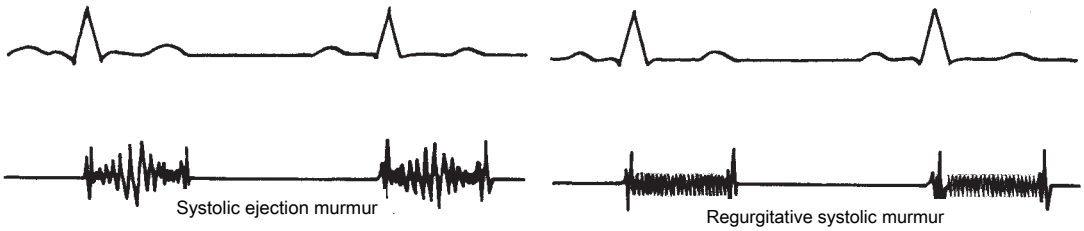


Fig. 41.1 Systolic murmurs.

There is a valve injury itself. It is more frequent than the murmur of organic mitral insufficiency.

semiography

Intensity. It is generally between grades 3 and 5.

Tone. Variable, although not as acute as that of mitral regurgitation.

Ring. Variable.

Duration. It is usually a holosystolic murmur.

Spread. Very weak towards the axillary line.

Modifications. It varies according to the response of the myocardium to treatment with cardiotonics; In some cases it disappears completely only to reappear when a new decompensation occurs.

BA A PART SYSTOLIC MURPUTS
OF THE STERNUM OR THE XIFHOID APPENDIX OR
TRICUSPID

Tricuspid regurgitation murmur

Semiogenesis or pathophysiology

It is caused by an injury to the tricuspid valve, generally of rheumatic etiology, or in the course of bacterial endocarditis.

Semiography

Intensity. It is generally a grade 2 or 3 murmur.

Tone. Sharp, although not as sharp as mitral regurgitation murmurs.

Ring. Blower, which can become harsh on some occasions.

Zions.

Moment of cardiac revolution. Systole.

Duration. It is a holosystolic murmur that begins with the first sound.

Highest intensity site. It is heard at the level of the xiphoid appendix, on the left side.

Spread. It spreads to the left to the anterior axillary line, and if it is very intense to the right of the sternum. In some cases it can be heard in the left paravertebral region.

Modifications. It varies with breathing, generally increasing in intensity with deep breathing. This modification is possibly due to the increase in ventricular filling that occurs during inspiration.

Tricuspid incompetence murmur

Semiogenesis or

It is the most common murmur at the level of the tricuspid focus. It is produced by dilation of the right chambers in patients with mitral stenosis or pulmonary artery hypertension.

The valve, although it does not have an organic lesion, cannot completely close the tricuspid orifice.

Semiography

Intensity. It is a murmur of low intensity, generally grade 1 6 2.

Tone. Low.

Ring. Gentle.

Location. Systole.

Duration. Holosystolic.

Site of highest intensity. Tricuspid focus.

Modifications. It disappears when heart failure is compensated.

SYSTOLIC MURPUTS FROM THE BASE, TO THE LEFT OF THE STERNUM

Organic pulmonary stenosis murmur

Semiogenesis or pathophysiology

It is almost always due to congenital heart disease, but it can be acquired due to endocarditis or infectious valvulitis of the pulmonary sigmoid valve.

Semiography

Intensity. It is one of the murmurs that reaches the highest intensity, almost always grade 4 6 5; It is only surpassed by the murmur of aortic stenosis.

Tone. Sharp.

Ring. Rough, scratchy.

Location. Systole.

Duration. It is a holosystolic murmur.

Highest intensity site. Pulmonary focus and second and third left intercostal spaces next to the sternum.

Spread. It has a wide spread, mainly towards the left sternoclavicular joint; When its intensity is very great, it can spread to the entire precordial region and in some cases to the highest part of the left scapulovertebral space.

Murmur of patent ductus arteriosus

Semiogenesis or physiopathology

As its name indicates, it is due to the maintenance of fetal communication between the aorta and the pulmonary artery after birth. Many authors consider that its presence during the first year of life may be normal.

semiography

Intensity. It varies between grades 3 and 4.

Tone. Sharp.

Ring. Raspy or like the noise of machinery.

Location. It is a continuous murmur, that is, it occupies systole and diastole with systolic reinforcement. Sometimes there is only a systolic murmur.

Duration. Holosistodiastolic; sometimes holosystolic only.

Highest intensity site. It is listened to in the first and second left intercostal spaces, 2-3 cm lateral to the left edge of the sternum.

Spread. When the murmur is intense, its systolic component spreads better than the diastolic component; It can reach the area of the tip as it spreads downwards and on the back it can be heard at the level of the left shoulder plate.

Modifications. The intensity increases with exercise and disappears after correct surgical treatment of patent ductus arteriosus.

accidental blow

It was described in Chapter 11 of Section I.

Murmur of relative pulmonary stenosis

Semiogenesis or

In this case it is not an injury to the sigmoid valve, but rather an injury to the pulmonary artery accompanied by an increase in the diameter of said artery, which

It results in the diameter of the valve orifice being narrow in relation to the arterial diameter. When is the diameter of the pulmonary artery increased? Under two main conditions:

1. When there is a dynamic increase, that is, when pulmonary arterial pressure is increased (mitral stenosis, chronic bronchopleuropulmonary lesions).
2. When there is an alteration of the arterial wall as in cases of sclerosis of the pulmonary artery.

Semiography

Intensity. Grade 1 or 2.

Tone. Acute, but less than in organic stenosis.

Ring. Gentle.

Location. Systolic murmur.

Duration. Holosystolic.

Highest intensity site. Lung focus.

Spread. Little or none.

Modifications. He doesn't have it.

SYSTOLIC MURPUTS FROM THE BASE, TO THE RIGHT OF THE STERNUM

Murmurs of organic aortic stenosis

Semiogenesis or

This murmur is due to lesions of the aortic valve that reduce the diameter of the valve orifice, resulting in rupture of the fluid column and turbulence of the blood flow.

Aortic stenosis can be congenital or acquired. The latter is due to lesions of rheumatic etiology in young individuals and valvular sclerosis in the elderly. It can also appear during the course of infectious bacterial endocarditis.

semiography

Intensity. It is one of the most intense murmurs, if not the most intense. Reaches grades 4, 5 and 6.

Tone. Sharp.

Ring. It is scratchy, although in some cases it can be musical.

Location. Systole.

Duration. Holosystolic.

Highest intensity site. Aortic focus.

Spread. It spreads to the right, upward to the right sternoclavicular joint and to the

neck vessels. In some cases it is so intense that it is heard throughout the precordial region; It can be heard without needing to apply the stethoscope to the chest wall.

Modifications. He doesn't have it.

The second heart sound may completely disappear from the right second intercostal space in severe aortic stenosis; In less advanced stages it may be decreased or normal. The masking of the second sound at the base can be recognized on auscultation, if a relatively obvious second sound is heard, which is due to the valve maintaining a certain degree of flexibility. The second sound, heard in the right second intercostal space, may be due to a reinforced pulmonary component. When the two components of the second noise are heard, a normal splitting or a paradoxical splitting can occur."

Differential diagnosis

The differential diagnosis of this murmur with respect to other systolic murmurs associated with hypertension and arteriosclerosis, which are also heard in the basal foci, will be discussed later. The diagnosis, which is quite easy when it is a murmur of isolated mitral regurgitation, becomes more complicated if, in the presence of a murmur of aortic stenosis, one wishes to confirm the existence of another murmur of mitral regurgitation. The aortic stenosis murmur propagates well toward the apex region, while the mitral regurgitation murmur propagates poorly toward the aortic focus. The presence of a murmur of similar timbre in both regions generally leaves no room for doubt that it is a murmur of aortic stenosis; However, it may be impossible to determine by auscultation whether additional mitral regurgitation also exists. In the presence of a not very intense aortic murmur, the finding of two murmurs of different tones will allow us to assume that both lesions probably exist.

Relative aortic stenosis murmur

The basal systolic murmur, associated with other signs of arteriosclerosis, accompanied or not by arterial hypertension, is the one heard most frequently in elderly people.

Semiography

Intensity. It is not very intense, grade 1 or 2.

Tone. Serious or medium.

Ring. Soft, although in some cases it can be scratchy or rough.

Location. Systolic.

Duration. Holosystolic.

Highest intensity site. Aortic focus.

Spread. Right sternoclavicular joint and neck vessels.

Semiogenesis or pathophysiology

Arteriosclerotic discoloration of the aorta and its valves and relative aortic stenosis, which results from the dilation of this artery, are factors that can lead to the appearance of this murmur.

When the murmur of aortic stenosis is intense and harsh, there is no possibility of confusing it with the murmur of relative aortic stenosis. If it is of moderate intensity, it can be differentiated with the help of the following data:

1. They generally occur at different ages, although calcific aortic stenosis appears in patients of approximately the same age.
2. The presence of a dilated aorta, hypertension or arteriosclerosis, leads to the diagnosis of relative aortic stenosis. The presence of other murmurs (indicating aortic insufficiency or mitral stenosis) favors the diagnosis of organic aortic stenosis.
3. In patients with arteriosclerosis or hypertension, the second sound is normal or enhanced, while it is absent or decreased in patients with aortic stenosis.

MESOCARDIAL SYSTOLIC MURPUTS

We will only describe the murmur of ventricular septal defect or Roger's disease and the murmur produced by an aberrant chorda tendineae.

Murmur of ventricular septal defect Roger's disease

Semiogenesis or pathophysiology

This murmur appears due to the existence of a hole in the interventricular septum, through this communication blood passes from the left ventricle to the right ventricle during systole, since the pressure inside the left ventricle is more than 120 mm Hg, while the pressure in the right ventricle does not exceed 30 mm Hg. It is a holosystolic regurgitation murmur.

Semiography

Intensity. Reaches grade 4 6 5 intensity.

Tone. Sharp.

Ring. Gritty.

Location. In systole, as it is the moment when blood passes through the interventricular orifice.

Duration. Holosystolic.

Highest intensity site. Third or fourth left intercostal space next to the sternum.

Spread. It has a large spread in the form of a transverse bar in the precordial region.

If the malformation is not very large, heart sounds are preserved. Loud murmurs may mask the aortic component of the second sound. If the malformation is evident, there may be strengthening of the pulmonary component. The splitting of the second sound is normal.

When there is enlargement of the left ventricle due to the malformation, a middiastolic murmur may appear.

Aberrant tendon chord murmur

Semiogenesis or pathophysiology

This murmur appears when there is a loose pillar, rupture of a chorda tendinous, or when there is a vibrating tongue that remains floating in the bloodstream when a leaflet ruptures.

Semiography

Intensity. It varies from grade 3 to grade 5.

Tone. Sharp.

Ring. Musical on guimbarda or piante.

Location. In systole, because it is the moment of greatest speed of the blood flow.

Duration. Holosystolic.

Highest intensity site. Generally it is in the mesocardium, but it can vary according to the position occupied by the valve tongue, or the chord that is broken, and this depends on the anatomical conditions of the ventricle.

Spread. It spreads eccentrically from the center or point of greatest intensity to the periphery.

Modifications. He doesn't have it.

DIASTOLIC Murmurs

Diastolic murmurs, as their name indicates, are those found in diastole, that is, between the second and first sounds. They can be of three kinds:

1. Decrescendo diastolic murmur, which begins immediately after the second sound. It is called protodiastolic or arterial diastolic.
2. Diastolic murmur that begins in middiastole, also referred to as passive atrioventricular diastolic.
3. End-diastolic murmur, also called presystolic or atriosystolic

The arterial diastolic murmur begins immediately after valve closure. Actually, more than a decrescendo, it is a crescendo-decrescendo, since it increases in intensity, reaches the acmé and begins to decrease.

The aortic arterial diastolic murmur is often heard at Erb's point (left third intercostal space).

It results from aortic and pulmonary regurgitation.

The passive atrioventricular diastolic murmur accompanies relative or absolute stenosis of the mitral or tricuspid valve. It is always separated from the second noise by a small interval.

The atriosystolic murmur, better termed presystolic, since it is essentially an ejection murmur and is often heard only in cases of minimal mitral stenosis; It is understood that in these cases, when atrial systole occurs, there will be sufficient atrioventricular blood flow to produce the murmur.

DIASTOLIC TIP RUMPS Murmur of

organic mitral stenosis

Semiography

Intensity. It is not a very intense murmur. It has variations: its intensity increases during the rapid filling phase, followed by a slight decrease and another increase that corresponds to the contraction phase or atrial systole. When there is no atrial systole, this presystolic reinforcement does not occur.

Tone. Serious.

Ring. It is heard in the form of a rolling noise or noise, hence its French name roulement.

Location. In diastole.

Duration. Holodiastolic.

Highest intensity site. It is heard in the mitral focus at the level of the apex of the heart.

Spread. Sometimes it spreads to the mesocardial area.

Modifications. They are produced by alterations in atrial contraction. This murmur can be modified with changes in position.

It is best heard with the patient in the left lateral decubitus position and may disappear in the standing position. He

Exercise modifies the intensity of the murmur and favors auscultation. In cases where it cannot be heard with the patient at rest, inhalation of amyl nitrite is used to reveal a murmur that was not auscultated under baseline conditions (French school).

Mitral stenosis is usually associated with an intense, bright first sound, which has been called the first crackle sound. In some cases, a mitral opening click is associated, which can be confused with a splitting of the second heart sound at the apex. These acoustic phenomena were described by Duroziez with the following onomatopoeia: ffut-ta-ta-rrou where ut represents the first sound, ta-ta the opening click of the mitral valve, rrou the roulement or diastolic dragging and ffel reinforcement or presystolic murmur.

The second sound may be reinforced in the pulmonary focus, but this is a late onset event and is due to increased pressure in the pulmonary circulation.

Relationship between the intensity of the murmur and the degree of injury

If you try to relate them, the following factors must be taken into consideration:

1. The presence of a thick chest wall or emphysema can reduce a loud murmur.
2. Cardiac minute volume must be taken into account, especially if it is measured as a function of heart rate. The increase in this, as occurs in excitement, exercise, thyrotoxicosis and fever, is associated with a considerable increase in the intensity of the murmur. At rest and in myxedema, the murmur decreases in intensity.
3. When the enlarged right ventricle displaces the left chambers posteriorly and outward, the murmur is heard very faintly in the midaxillary line, and may go unnoticed completely.
4. When the stenosis is very advanced, the caliber of the liquid column that gives rise to the murmur may be very small and, therefore, the noise produced is less evident.

Despite the existence of many factors capable of modifying the relationship between the intensity of the murmur and the degree of stenosis, this relationship remains constant in most cases.

Due attention should be paid to the duration of the murmur, since, like the intensity, it is closely related to the degree of stenosis; duration is subject, however, to fewer modification factors.

Flint relative or functional mitral stenosis murmur

Semiogenesis or pathophysiology

It is a murmur that is due to a functional or relative narrowness, which is produced by an enlargement of the left ventricle. In these cases, the mitral valve is not injured, but the left ventricle's enlargement results in the rupture of the liquid column at its exit from the orifice of the mitral valve, which results in the appearance of a murmur or diastolic dragging noise.

As this murmur is heard more frequently in cases of aortic insufficiency, some authors have attributed its mechanism of production to a true stenosis of the mitral valve, although always of a functional nature. These authors consider that the mitral valve, when displaced by the flow of blood that refluxes from the aorta towards the left ventricle, produces a narrowing of the mitral valve with the appearance of a diastolic murmur with the characteristics of a roulenent.

semiography

The characteristics of this murmur are the same as those of the murmur of organic mitral stenosis. The differential diagnosis is based on the fact that its main component is made up of high and irregular vibrations that begin in the final part of diastole and are not preceded by the mitral opening click.

DIASTOLIC RUMBERS OF THE BA A PART OF THE STERNUM OR THE XYPHOID APPENDIX

Tricuspid stenosis murmur

In general, the diastolic murmur of tricuspid stenosis has the same location and virtually the same pitch and timbre as the murmur of mitral stenosis.
However, on certain occasions it may have a higher pitch and occur earlier in diastole, somewhat resembling the protodiastolic murmur of aortic and pulmonary insufficiency. It is best heard sitting down, with the bell receiver. Reinforcement of the first sound is less frequent than in mitral stenosis.

Tricuspid stenosis is almost always accompanied by mitral valve disease, the symptoms of which can overlap with the first.

However, generally, the tricuspid stenosis murmur can be recognized even in the presence of mitral stenosis, due to its characteristic location and respiratory modifications. This murmur is best heard immediately to the left of the xiphoid process and in the left fourth intercostal space, near the sternum; It is generally well localized, without extending much to the left, even in cases where it is intense and there is enlargement of the right cavities.

When murmurs of mitral and tricuspid stenosis occur simultaneously, two auscultation foci can frequently be determined, one near the sternum and the other at the tip, which can deviate to the left in case of cardiac enlargement.

As with the murmur of tricuspid regurgitation, the murmur of stenosis increases markedly in intensity during inspiration. On certain occasions it can be of equal intensity in both phases of breathing, or it can be heard only during inspiration. On the other hand, the murmur of mitral stenosis may remain unchanged, or decrease in intensity during inspiration.

The presence of a palpable venous beat in the jugular, synchronous with the presystolic phase of the murmur, can be a valuable sign to confirm the diagnosis.

DIASTOLIC Murmurs at the base, to the left of the sternum

Murmur of organic aortic insufficiency

Semiogenesis or

The diastolic murmur of aortic insufficiency is generally due to a rheumatic heart lesion and increasingly less frequently due to syphilitic aortitis. Low intensity murmurs may also occur in hypertension and sometimes in heart disease of arteriosclerotic etiology. The sudden appearance of a murmur of this type in a hypertensive patient should suggest a dissecting aneurysm. Some congenital heart diseases, such as bicuspid aortic valve, can be the cause of aortic insufficiency.

semiography

Intensity. It is a murmur that reaches grade 2 6 3 intensity.

Tone. Serious.

Ring. Smooth, aspirational, rarely scratchy.

Location. In diastole.

Highest intensity site. Left third intercostal space, next to the edge of the sternum. At the site known as Erb's second aortic focus,

Duration. Holodiastolic.

Spread. Left edge of the sternum, towards the tip.

Modifications. He doesn't have it.

Murmur of pulmonary insufficiency Gra am
Steell murmur

semiography

This murmur has the same location, timbre, and pitch as that of aortic insufficiency. Its focus of auscultation is in the second or third left intercostal space. If it is severe enough, it spreads to the left edge of the sternum. As it is a high-pitched murmur, the diaphragm receiver should be preferred to auscultate it, and it is necessary

Focus attention on the period immediately following the second sound, since the accentuated pulmonary component of this noise masks the murmur. Generally there is a reinforcement of the second sound, due to existing pulmonary hypertension.

Semiogenesis or pathophysiology

Pulmonary artery insufficiency is generally associated with pulmonary hypertension and/or dilation of the pulmonary valve annulus, although occasionally it may be due to rheumatic fever, congenital lesions, or an aneurysm. Any condition capable of producing pulmonary hypertension can give rise to the production of this murmur: mitral stenosis, right heart failure, idiopathic or primitive pulmonary hypertension and pulmonary hypertension associated with congenital heart lesions.

The differential diagnosis between the murmur of pulmonary insufficiency and that of aortic insufficiency may present difficulties in cases in which the peripheral signs of aortic insufficiency are not well defined.

In these cases the following data may be valuable:

1. In the case of other signs of rheumatic heart damage, it is more correct to assume that the murmur is due to mild aortic insufficiency, despite the existence of obvious signs in favor of pulmonary hypertension.

2. The presence of a systolic murmur of aortic stenosis can be considered as evidence that the murmur under study is due to aortic insufficiency.

3. If there is an exaggerated pulmonary component and there are no signs of rheumatic cardiac damage, it can be assumed that the murmur originates from pulmonary insufficiency.

4. An intense murmur, in the absence of peripheral signs of aortic insufficiency, favors the diagnosis of pulmonary insufficiency.

5. If there is wide spread, it is aortic insufficiency: the murmur of pulmonary insufficiency is rarely heard in the right second intercostal space.

6. The murmur of aortic insufficiency is usually louder on expiration.

7. If the insufficiency is at an advanced stage of evolution, the data obtained with fluoroscopy can be valuable, since it indicates which of the two arteries shows an increase in the pulsatile wave. The verification of a dilated pulmonary artery favors the diagnosis of pulmonary insufficiency.

Murmur of relative aortic incompetence

Continuing with the diastolic murmurs on the left of the sternum, we find that of aortic incompetence.

Functional insufficiency of the aortic sigmoids is

An extremely rare occurrence, it appears in sudden increases in blood pressure. The murmur of this insufficiency is similar to the murmur of organic aortic insufficiency, but less intense (grade 2).

Diastolic murmurs at the base, to the right of the sternum, such as the diastolic murmur of organic aortic insufficiency and the murmur of relative aortic incompetence, have already been described when studying diastolic murmurs at the base, to the left of the sternum.

To conclude the chapter, we will explain other aspects of auscultation such as venous rumor, pericardial rub, and extracardiac auscultation.

VENOUS RUMOR

It is a continuous, low-pitched rumble heard in the neck and upper chest of many children and some adults. Its site of greatest intensity is generally above the clavicle, in the angle between it and the insertion of the sternocleidomastoid. On some occasions, however, it can be heard at the base of the heart and, very rarely, extending across the breastbone. The rumor is heard on both the left and the right, but is more common on the right.

It is best perceived with the patient sitting and especially if he turns his head to the left with his chin raised. Normal breathing generally does not modify its intensity, or it may accentuate it during inspiration.

The rumor is due to the formation of eddies in the bloodstream of the internal jugular vein, possibly at the point where it joins the brachiocephalic trunk. Therefore, it can be interrupted by pressing the finger on the internal jugular vein, at the level of the thyroid cartilage, between the trachea and the sternocleidomastoid.

Its appearance is conditioned by factors that increase blood flow, for example, thyrotoxicosis and anemia.

When the murmur is heard well at the base of the heart, it should be differentiated from the murmur of patent ductus arteriosus.

The best element of judgment, in these cases, is to remember the possibility of its appearance; It is then easily identified and can be interrupted by applying pressure to the neck.

PERIC RDICO RUB

Pericardial rubbing noise results from the friction of the swollen and "glossy" pericardial sheets. can go accompanied by a palpatory thrill. It is heard in the precordium, near the base of the heart. It differs from valve tones and murmurs by its acoustic character of friction, which is reminiscent of leather or new bills when folded; by auscultation independently of heart sounds; for not spreading; due to its variable character from one examination to another: due to increasing intensity when leaning the body forward, during forced expiration or when applying pressure with the diaphragm of the stethoscope. It is distinguished from pleural friction rub by the fact that the latter disappears during apnea.

Below we present a summary of the murmurs studied:

EXTRACARDIAC AUSCULTATION

Auscultation of arteries and veins outside the chest can provide valuable data. The venous rumble has already been described, which can be heard in the neck and sometimes in the precordial region. In patients with thyrotoxicosis, a continuous murmur over the thyroid gland can sometimes be heard, produced by the marked increase in its irrigation. In patients with cirrhosis and portal hypertension, a continuous, high-pitched rumble can be heard near the xiphoid appendix and the epigastric region. This venous rumor is due to the formation of anastomoses between the portal system and the general circulation, especially at the level of the paraumbilical veins. Rarely, a venous murmur can be heard over an enlarged spleen.

The continuous murmur heard in patent ductus arteriosus is a clear example of murmurs that They are heard whenever there is a fistula or an arteriovenous aneurysm. At the site of the injury, a continuous murmur with systolic reinforcement accompanied by a thrill is produced.

Under normal conditions, murmurs are not perceived at the level of the arteries; However, compression of a large-caliber artery allows a murmur to be heard that is synchronous with the pulse wave. This murmur is due to the eddies that form in the bloodstream, at the site where the pressure is exerted. In aortic insufficiency and other conditions characterized by increased pulse wave and blood flow velocity, the murmur produced by compression is more intense, and it is often possible to hear two murmurs: one systolic and one diastolic. This double murmur heard in the femoral artery is known as Duroziez's sign; it is observed in aortic insufficiency and thyrotoxicosis and, occasionally, in hypertension and fever.

42

COMPLEMENTARY EX MENES IN CARDIOVASCULAR CONDITIONS

Among the complementary tests that are widely used in cardiovascular conditions, we have:

- Laboratory exams.
- Volemia.
- Assessment of cardiac work.
- Cardiac catheterization.
- Pericardial puncture.
- Radiographic study of the heart.
- Electrocardiography.
- Stress test (Master's test).
- Phonocardiography.
- Computed axial tomography
- Nuclear magnetic resonance.
- Positron emission tomography.
- Perfusion study with isotopes and stress echocardiography.
- Ergometric tests.

With the exception of the electrocardiogram and the stress test, or Master's test, which will be treated in separate chapters, the rest of the exams will be explained below with a purely elementary approach.

EX LABORATORY MENES

In this group we will refer to the tests that may have a semiological value in cardiovascular patients, such as:

erythrosedimentation

It is very useful in those patients who suffer from rheumatic or coronary heart disease.

In rheumatic heart diseases, erythrocyte sedimentation rate is very useful to determine the activity of the disease. At the moment they begin, the erythrocyte sedimentation rate is not modified, but it takes a few days for the acceleration to occur, increasing until it reaches a maximum figure and slowly decreasing as the activity of the process subsides.

In bacterial endocarditis, both acute and subacute, there is an increase in the erythrocyte sedimentation rate that reaches more than 40 mm in the first hour, with an average of 72 mm. Variations in erythrocyte sedimentation rate can serve as a guide to determine the effectiveness of antibiotic treatment in this disease.

In myocardial infarction due to coronary occlusion, erythrocyte sedimentation takes approximately one week to accelerate. During the healing process, the acceleration of erythrocyte sedimentation rate slowly decreases until it becomes normal, although in some cases it may remain slightly elevated for some time.

EMOGRAM

In cardiovascular diseases, alterations may occur in both the white series and the red series of the hemocytogram.

Alterations of the white series

In active rheumatic heart disease, a leukocytosis is Goes observed that can reach up to 12,000-15,000 leukocytes per cubic millimeter with an increase in neutrophils. This leukocytosis occurs before the elevation of the erythrocyte sedimentation rate occurs. When the rheumatic fever outbreak ends, neutrophilia is replaced by lymphocytosis and eosinophilia. In bacterial endocarditis, leukocytosis is also observed, which can reach up to 20,000 leukocytes per cubic millimeter with a neutrophilia of 95%.

Myocardial infarction due to coronary occlusion is accompanied by mild leukocytosis with neutrophilia, except in cases of very extensive infarction in which the alterations are greater, and in small infarctions that can evolve without alterations in the leukocyte formula.

Pulmonary infarcts produce leukocytosis with neutrophilia.

Alterations of the red series

In bacterial endocarditis the number of erythrocytes ranges between 2 and 4 million per cubic millimeter and the hemoglobin level drops to 40 6 70%. In the terminal period of the disease the anemia is more intense.

Moderate anemias are found in patients with active rheumatic heart disease, with numbers rarely lower than 3.5 million per cubic millimeter of erythrocytes.

The anemia of infectious heart diseases, both of rheumatic and bacterial etiology, is of the hypochromic microcytic type.

In malignant hypertension with nephrosclerosis and renal failure, intense anemias are found accompanied by medullary aplasia produced by toxic substances.

cas originating in the kidney or due to a deficiency in the production of erythropoietin, by it.

In heart disease with anoxia and cyanosis, an increase in the number of erythrocytes occurs; This increase (polyglobulia) can reach up to 8 million red blood cells per cubic millimeter, or more. These alterations occur in cyanotic congenital heart disease and chronic cor pulmonale.

SEROLOGICAL REACTIONS

These investigations should be indicated to all patients with heart disease, especially those who have an aortic lesion to determine the possible syphilitic etiology.

PROTHROMBIN TIME

It is the minimum time it takes for citrated or oxalated blood to clot when optimal amounts of thromboplastin and calcium are added. In normal subjects it is 12-15 s. This test is very useful in the study of myocardial infarction, pulmonary infarction, peripheral arteriopathies and vein diseases. It is an essential determination to maintain therapy with anticoagulants.

EMOCULTURE

Blood culture, whether venous or arterial, is very useful in the diagnosis of bacterial endocarditis. The negativity or positivity of a single blood culture has no diagnostic value. It is necessary to do serial cultures and obtain two to four positive cultures to make the definitive diagnosis. Some researchers have obtained better results using blood obtained by puncture of the bone marrow for cultures (myeloculture).

BLOOD LIPIDS

There is overwhelming evidence that one of the most important risk factors in atherosclerosis is elevated blood lipids. This is more important in young patients and decreases with age.

Elevated blood cholesterol level is associated with premature risk of extensive coronary artery disease and early myocardial infarction.

Elevated triglyceride levels also lead to an increased risk of myocardial infarction, whether with high or normal cholesterol. Elevation of triglycerides is frequently caused by carbohydrates and is associated with diabetes mellitus.

DETERMINATION OF IN SERUM IMAS

Among the serum enzymes useful in cardiovascular conditions are the following:

Glutamic oxalactic transaminase TGO

It is widely distributed in the human body; Its activity is maximum in the cardiac muscle, followed in order of importance by the skeletal muscle, the brain, the liver and the kidney. When tissue is destroyed or necrotic, the enzyme is released from the cell.
Due to its high concentration in the myocardium, when tissue necrosis occurs, the activity of TGO in the serum increases significantly. This assessment has been used to diagnose the existence of a myocardial infarction.

The normal activity of TGO in serum is considered to be 40 U/dL of serum, or also 15 UVdL. In cases of myocardial infarction, TGO activity in the serum rises above 40 U/dL between six and thirty-six hours following the infarction. Serial determinations must be made to help us verify whether there has been a progressive increase in the lesion. High transaminase values after a heart attack reach a peak and then return to normal within a week. In cases of liver injury, considerable elevations in transaminase values occur over a much longer period of time.

Lactic hydrogenase and its isoenzymes

Within this group, five fractions have been electrophoretically separated, which are distinguished by the different speeds in their electrophoretic mobility and their immunochemical activities.

Isoenzyme 5 of lactic dehydrogenase of the alpha 1 group, with rapid mobility, is the one that usually corresponds to the increased values in myocardial infarction. Normal values are 100-300 U. When it increases due to a pathological process, it does so in the first 12-24 hours, reaching the maximum level between 2 and 4 days later, to gradually return to normal. It begins later than OGT and also normalizes after it.

Creatine phospho uinase

Serial determination of creatine kinase or creatine phosphokinase (CK) is useful in the diagnosis of acute myocardial infarction. Normal values are £235 U/L (man) and £ 190 U/L (woman), it rises in the blood 6 hours after myocardial necrosis and remains elevated for 36-48 hours. The myocardial fraction is the MB (<3% of the total). CK elevations with more than 40% MB are diagnostic, when accompanied by clinical findings suggestive of infarction.

The infarcted myocardium also releases myoglobin and the contractile proteins troponin-T and troponin-I. Is-

The latter seem to be very sensitive indicators of myocardial injury and can replace the usual CK-MB analysis, at the beginning, when pain and the ECG are not diagnostic.

URINE

In patients with heart disease, and particularly when the existence of heart failure is suspected, the urine eliminated for 24 hours must be collected, which allows us to establish a balance between the amount of fluid ingested and the fluid eliminated.

The most important data from the urine test are:
density, albuminuria, glycosuria and sediment.

The study of density is important to determine the state of kidney function. It is completed by dilution and concentration tests.

In patients with high blood pressure, along with the urine test, a descending pyelography and other imaging tests should be performed to study the kidneys and adrenals.

VITAL CAPACITY CV

The determination of vital capacity is done using the spirometer; It is a test that can be modified due to lack of operator training, lung diseases and cardiac disorders. If we can exclude the first two factors we have to accept that the modification of the VC is due to the state of congestion within the pulmonary circulation. The CV in the normal individual ranges between 2 and 5 L, and varies according to age, sex, height and weight.

CAPTURE OF I

It is indicated when there is the possibility of a cardiac disorder caused by a thyroid disease, either due to its hyperfunction or hypofunction.

VOLEMIA

The techniques currently used to determine the amount of circulating blood (volemia) are not very exact, but since the clinical variations are much greater than the margin of error, results of practical interest can be obtained.

These techniques are based on the intravenous injection of a dye in a colloidal state that diffuses rapidly into the plasma, but is not absorbed by the tissues.
If doses of the dye are made in the venous blood at regular intervals, we can determine the amount of circulating blood.

The most frequently used dye is Evans blue and the technique used is as follows:

a) A venous blood sample is obtained without using a ligature or tourniquet and the plasma blood cell ratio is determined by the hematocrit.

b) 10 mL of physiological saline solution containing Evans blue is injected into an antecubital vein, without using a ligature or tourniquet, according to the patient's weight. The time is noted.

c) Blood samples are obtained from the opposite vein 10 minutes after injecting the dye and these samples are compared with a standard using a colorimeter. In normal individuals, complete mixing of the dye with the blood is complete 9 min after injection.

In cases of heart failure, it can last up to 15 minutes or more.

Normal individuals have an average of 77.5 mL of blood per kilogram of weight in men and 66 mL per kilogram of weight in women. The accumulation of blood in the organs that serve as reservoirs and slow circulation in the capillary areas can produce a marked variation in the amount of circulating blood. When the amount of circulating blood is increased, it is said that there is plethora.

ASSESSMENT OF CARDIAC WORK

In this aspect the heart can be considered from three points of view:

-As a propellant pump: hemodynamics.

-Like muscle.

- As a component of circulation.

Hemodynamic measurements are used to determine its function as a delivery pump.

To check its function as a muscle, the principles of muscle mechanics are applied. The function of the heart as a component of circulation is reflected in disorders resulting from low cardiac output, insufficient organ perfusion, and congestion.

venous.

THE HEART AS AN EMODIN MICA IMPELLENT

cardiac output

Indicator dilution techniques are mainly used to determine cardiac output, without removing the patient from their room. In adults, normal limits at rest range between 2.5 and 3.6 L per minute per square meter of body surface. A decrease in resting output represents a late stage of abnormal cardiac work, since the lack of increase in cardiac output during exercise begins much earlier.

During exercise, in normal hearts the heart rate, and consequently the output, increases as do the linear functions of oxygen consumption. These increases in cardiac output are achieved mainly by acceleration of the heart rather than by increase in stroke volume.

In cases of heart failure, this expenditure depends even more on the frequency, both at rest and during exercise.

THE HEART AS M SCUL0

Contractility, relaxation and distention, and energy metabolism of the myocardium are measured using different techniques (rate of increase in intraventricular pressure, speed of contraction, etc.), often cumbersome and traumatic; Today they are in the experimental period and cannot yet be used in hospitalization wards.

THE HEART AS A COMPONENT OF THE CIRCULATION

When an overloaded ventricle fails, two consequences occur:

1. Venous hypertension.

2. Slowing down of circulation.

Both can be measured without moving the patient from their room.

circulatory speed

Circulation time is studied by injecting a chemical substance into the vein that will produce its effects after it has passed through a certain part of the circulatory system.

In practice three different types of tests are used:

- Arm-to-periphery circulation time.

- Arm-to-lung circulation time.

- Lung to periphery circulation time.

Arm to periphery circulation time

The most used of these tests is the determination of arm-to-tongue circulation time, in which calcium gluconate is used. To do this, 5 mL of a 10% calcium gluconate solution is injected into one of the superficial veins of the forearm in a time no longer than 3 s. The arrival of the drug on the tongue is noticed by the patient because it produces a sensation of heat on the tongue. The normal arm-to-tongue circulation time ranges between 14-18 s. It is prolonged in heart failure.

Arm-to-lung circulation time

For this test, 2 mL of ether and ½ m of physiological saline solution are quickly injected. The action of

ether on respiration when vapors appear in the exhaled air is noted by both the patient and the researcher.
The normal arm-to-lung circulation time is short, averaging 4-8 s.

Lung to periphery circulation time

It is the arithmetic difference between the arm-to-periphery time and the arm-to-lung time; The normal circulation time is approximately 7-1l1 s.

CARDIAC CATHETERIZATION

This procedure allows:

1. Record and measure intracardiac and intravascular pressures.
2. Obtain blood samples from the different cavities to determine the oxygen and Co content,
3. Injection of contrast media for selective angiocardiography, as well as radioactive substances or dyes for the study of dilution curves.

RIGHT HEART CATHETERIZATION

This method is based on the introduction of a fine catheter into one of the superficial veins of the forearm. The catheter is taken to the heart through the venous system. It must be radiopaque, with a hole at the tip and a length of 100-125 cm. The introduction is done under the fluoroscopic screen to determine the progress of the catheter through the circulatory system.

With this method we can carry out different investigations that include intracardiac pressure determinations, blood gas studies and intracardiac electrocardiography.

Cardiomanometry

By means of a manometer connected to the catheter, graphic curves of pressure variations in the superior and inferior vena cava, right atrium, right ventricle, pulmonary artery can be obtained, and even the pressures in the pulmonary capillaries can be obtained.

Right intraventricular curve

This graph presents a plateau curve that is typical of intraventricular pressure graphs. A small positive curve marks the final filling produced by atrial contraction. The rapid rise curve corresponds to the isometric contraction phase; this curve ends in a small notch determined by the closure of the tricuspid valve. After further elevation, a second notch marks the opening of the pulmonary sigmoid valves. During the expulsion period

A plateau line is inscribed followed by a sharp drop that marks the end of the expulsion period. This is followed by two vibrations: the first marks the closure of the pulmonary sigmoid valves; the second, the opening of the tricuspid valve. The period of rapid filling is not accompanied by an increase in intraventricular pressure, demonstrating that the right ventricle is gradually distended by blood entering through the triscuspid valve. Intraventricular pressure ranges from +20 to +25 mm Hg during systole; to 0 during diastole.

Right intra-atrial curve

This graph presents a small positive wave produced by atrial contraction during presystole, followed by a depression (systolic collapse) that corresponds to the contraction of the ventricle. Intra-atrial pressure increases during the final part of systole and reaches its maximum at the moment of opening of the tricuspid valve.
There is then a progressive decrease in pressure until the next atrial contraction takes place. The pressure varies between 0 and 3 mm Hg.

Pulmonary artery curve

It is a typical arterial graph. The anachrotic notch is clearly visible; the incision, deep; the dicrotic wave, well marked, and the maximum point of the curve is rounded.
Pressures vary between +20 and +35 mm Hg during systole; between +5 and +8 mm Hg in diastole.

Curve of the pulmonary capillaries

This is essentially a venous graph whose curves are due to pressure changes in the pulmonary veins transmitted through the capillaries of the lung. The curve presents a positive atrial wave, a decrease during the first part of systole followed by a systolic collapse and an increase during the first phase of diastole, ending in a mid-diastolic collapse. The differences between this graph and that of the right atrium are the following: a higher mean pressure, a wave delay of approximately 0.08 s, and a deeper diastolic collapse. The average pressure is around +3 mm Hg.

Through cardiomanometry the following determinations can be made:

1. Mitral valve area.
2. Area of regurgitation or mitral insufficiency.
3. Determination of the amount of blood regurgitated through the area of mitral regurgitation.

Oximetry

It is the determination of the oxygen content and hemoglobin saturation in blood samples obtained.

nests through the catheter. These data are used to determine the minute volume, as well as the existence and size of an abnormal communication between the arterial and venous systems0.

The study of normal individuals has shown that the difference in oxygen content between the superior vena cava and the right atrium is 2.3% by volume;
between the right atrium and the right ventricle, 1.8% volume, and between the right ventricle and the pulmonary artery, 0.5% volume. Therefore, if a difference greater than those previously indicated is found, it indicates the existence of a communication through which oxygenated blood passes to the right cavity.

CORA ÓNI UIERDO CATHETERIZATION

OR THE AORTA

Aortic catheterization has been used to measure pulse pressure. The aortic pulse curve in man is similar to the curve obtained in the dog. Left atrial catheterization has been achieved in cases of atrial septal defect by passing the catheter through the hole in the atrial septum. Left ventricular catheterization has been attempted in man, first in patients with aortic insufficiency and then in normal subjects. The technique is as follows: the left brachial artery is isolated, through which a trocar is introduced and through it a catheter is taken to the arch of the aorta and then through the aortic valve to the left ventricle. The pressure curve of the left ventricle is similar to that of the right ventricle. The systolic pressure is the same as that of the aorta and the diastolic pressure is 0.

PERIC RDICA PUNCTURE

Pericardial puncture can be performed to:

1. Confirm the diagnosis of pericardial effusion.
2. Determine the causative agent.
3. Relieve the compressive symptoms caused by the spill.
4. Introduce medications into the pericardial cavity.

PLACE TO PERFORM THE PERIC RDICA PUNCTURE

Performing the puncture requires a radiological study of the chest, which helps us determine the most favorable point for the puncture. The most frequent sites are:

1. The left fifth intercostal space, about 2 cm inside the left edge of the area of percusable dullness, this being the most common site.

   mind used. If there is easily detectable tip impingement, the needle or trocar should be inserted 162 cm outside of it.

2. The angle between the xiphoid process and the left costal margin. This is the recommended site when the spill is large; At this point we approach the lowest portion of the pericardial sac. It is also the point of choice when purulent effusion is suspected, since this way we avoid spreading it to neighboring structures such as the lung and pleura.

3. The right fourth intercostal space, 1 cm toward the midline of the right border of the area of percussable cardiac dullness. It is the area of choice when most of the effusion is sacculated on the right side.

4. The seventh or eighth left intercostal space in the posterior region of the thorax on the midcapular line, the patient's left arm must be elevated to separate the scapula. This is the preferred site when there is evidence of a large effusion with lung compression, and fluid cannot be obtained easily through the other sites. This entry route should not be used in cases where purulent effusions are suspected due to the danger of contaminating the pleura.

PERIC RDICA PUNCTURE TECHNIQUE

Pericardial puncture must be performed with all the aseptic precautions of surgical procedures. The site chosen for the introduction of the needle or trocar as well as the subcutaneous tissue of the region must be anesthetized with 2% novocaine solution. A 7-8 cm long, 18 or 16 gauge, bevel-cut needle should be used for puncture, connected directly or by means of a tube or probe to a 20 or 30 mL syringe. The needle or trocar must be introduced through the anesthetized area until it is felt to penetrate the resistant pericardial membrane. This occurs at a depth of 3-5 cm from the skin surface, when the site chosen is one of the anterior intercostal spaces.

When the needle or trocar is inserted through the left fifth intercostal space, it is directed inward and posteriorly toward the spine; When the site chosen is the left costoxiphoid angle, it should be directed backwards, upwards and slightly inwards; When the puncture is performed in the right fourth intercostal space, it should be directed backwards and towards the midline, towards the spinal column; When the route used is the posterior one, the needle or trocar is directed forward and towards the midline.

RADIOGRAPHIC STUDY OF THE HEART

In radiographic studies of the heart, it is projected as a shadow that stands out in the middle of both lung fields. No details can be discovered in the cardiac shadow, we can only examine its silhouette and position.

The application of radiographic study to the examination of the heart and great vessels provides important data regarding the size, shape, position and movements of these organs. This study must be related to all other examinations performed on the cardiovascular system; If this is not done, there is a danger that a small constitutional anomaly will be considered pathological.

The radiographic study includes:

Radioscopy.

- Teleradiography.
- X-rays in oblique position.
- Angiocardiography.
-Coronary angiography.

RADIOSCOPY

It allows the different cavities of the heart and the great vessels to be examined separately, and to study segmental alterations but above all their pulsatility.
In addition, it provides details of the skeleton, costophrenic angles, mobility and position of the hemidiaphragms, mediastinum and lungs.

We will perform it first in a frontal position, with the anterior part of the patient's tőrax on the screen; then in anterior, right and left oblique, and finally, in anteroposterior position with the subject facing away from the observer.

Classic fluoroscopes have been replaced by modern equipment with image intensifiers, which allow this examination to be performed with a minimum of milliamps and in full light, avoiding the risk of radiation to the patient and especially to the doctor.

It has the disadvantage of deforming and enlarging the cardiac image, since the X-ray focus is very close to the fluoroscopic screen, which must be taken into account for the interpretation of the size of the heart.

TELERADIOGRAPHY

It is an x-ray that is taken with the focus approximately 2 m away from the subject to avoid the distortion already mentioned. It is the most used radiological procedure in cardiology due to its simplicity and relative accuracy.

RADIOGRAPHS IN OBLIQUE POSITION

They allow the configuration of the heart to be studied with different incidences. Filling is generally done simultaneous treatment of the esophagus with barium, with the aim of checking the deformations that the vessels or cavities of the heart produce in this organ when they enlarge or change position.

ANGIOCARDIOGRAPHY

It allows visualization of the chambers of the heart and the great vessels by rapidly injecting iodinated substances into the bloodstream. With this method, a fairly clear contrast is obtained that facilitates the interpretation and diagnosis of congenital anomalies and malformations.

CORONARIOGRAPHY

The repletion of the root of the aorta or the catheterization of the ostium of the coronary arteries is performed using different types of catheters, through which the contrast is injected, filling the two coronary arteries and their branches.
It allows you to specify their status so you can check whether or not there is total or partial occlusion.

ELECTROCARDIOGRAPH

It will be studied in the next chapter.

STRESS TEST MASTER TEST

It is a very common diagnostic tool currently used to assess coronary reserve; It will be dealt with in Chapter 44.

PHONOCARDIOGRAPHY

It is the recording, by means of special equipment, of heart sounds.

The objective of phonocardiography is to obtain a graphic record of what is heard when auscultating the heart and to more accurately specify the characteristics of the heart sounds or murmurs. It can be done directly by recording the vibrations of a membrane that acts as an eardrum. However, the most used method is electrical. The equipment used works in the following way: the heart sounds are collected by means of a microphone placed in the auscultation foci, the potential variations are transmitted to an amplification equipment that transforms the sounds into electric current, which is received by a mirror galvanometer placed between the poles of an electromagnet: the flashes of the mirror are recorded on a photographic paper that runs at a uniform speed and at the same time the electrocardiogram and phlebogram.

The acoustic phenomena that occur in the heart are of low tone and low intensity, which is why some are not perceived by the ear. This perceives high-pitched noises more easily than low-pitched ones.

When a heart sound is followed by a murmur that increases in intensity, the human ear cannot separate the sounds. The human ear also makes errors when there is a rapid heart rate, accompanied by murmur noise.

As galloping noises are low frequency, the ear sometimes does not appreciate them.

Murmur noises have a frequency of 100-150 vibrations per minute. Gallop rhythms, 40-60 vibrations per minute. The first noise has 90-100 vibrations per minute.

The usefulness of the phonocardiogram is that through it we can identify the different noises, as well as determine at what time in the cardiac cycle they occur.

The first noise consists of 4-7 oscillations with a duration of 0.14-0.10 s. It corresponds to the QRS complex of the electrocardiogram.

It can be decomposed into three parts:

1. Initial vibrations (isometric contraction).
2. Main vibrations (expulsion phase).
3. Terminal vibrations.

The second sound in the phonocardiogram is made up of 4-6 wide vibrations of slightly higher frequency; It corresponds to the end of the T wave of the electrocardiogram.

Abnormal noises are recorded according to their timbre and intensity, and at the exact moment of the cardiac cycle in which they occur.

In summary, phonocardiography is useful, since it provides information on acoustic phenomena not perceptible to the human ear and visibly specifies the time of the cardiac revolution in which they occur.

Noises that appear indisputable by auscultation are shown to be inaccurate. When there are doubts or disparities in the interpretation of what is heard, the phonocardiogram allows the diagnosis to be established. However, the phonocardiogram cannot at any time replace auscultation, since it requires equipment and technicians that are not available to all hospital or polyclinic centers.

TRADITIONAL ULTRASONIC ECHOCARDIOGRAPHY AND DOPPLER ECHOCARDIOGRAPHY

Echocardiography for diagnostic purposes constitutes an important weapon in cardiology. It promotes the study of different pathologies of the pericardium and valves, as well as quantitatively evaluating the function of the ventricles. With Doppler echocardiography, vascular blood flow can be studied and allows us to distinguish important flow data in the study of cardiac valve hemodynamics.

VECTOCARDIOGRAPHY

Like electrocardiography, it is an instrumental method of graphic recording. Both are based on the same principle of orthogonal projections and correlation of various perspectives and pursue the same purpose: appreciation of the intensity, direction and direction of the successive moments of the special electromotive force generated by the heart during the cardiac cycle, that is, three-dimensional vector magnitudes represented by their symbol (the arrow or vector).

Despite the differences that exist between electrocardiography and vectocardiography graphs, both are based on the same principle and appreciate the same phenomenon.

The vectocardiogram (VCG) is the multidirectional projection of the cardiac electromotive force, in a plane made up of two perspectives or components.

The electrocardiogram is an analysis, however the vectocardiogram is a synthesis of the electromotive force of the myocardium and is a complement to the first; the latter can be flat or spatial.

ADA COMPUTER AXIAL TOMOGRAPHY

It is useful to evaluate aneurysms and thinning of the ventricular wall, calcification of the coronary arteries, etc.

NUCLEAR MAGNETIC RESONANCE

It is useful in ischemic heart disease to measure wall thinning and define residual myocardium in order to evaluate surgery and infarctions.

PERFUSION STUDIES WITH ISOTOPES AND STRESS ECHOCARDIOGRAPHY

At present there are controversies about the advantages and disadvantages of these investigations. Among the limitations of stress echocardiography are its deficiencies in the detection of multivessel disease, while studies with thallium and technesium have many false positives due to the superimposition of structures such as the breast and diaphragm.

ERGONOMIC TESTS

Over time they have replaced the Master's tests and have proven useful in the diagnosis of coronary heart disease.

43

BASICS OF ELECTROCARDIOGRAPHY. ELECTROCARDIOGRAPHIC

ELECTROPHYSIOLOGICAL FUNDAMENTALS OF THE ELECTROCARDI0GRAM

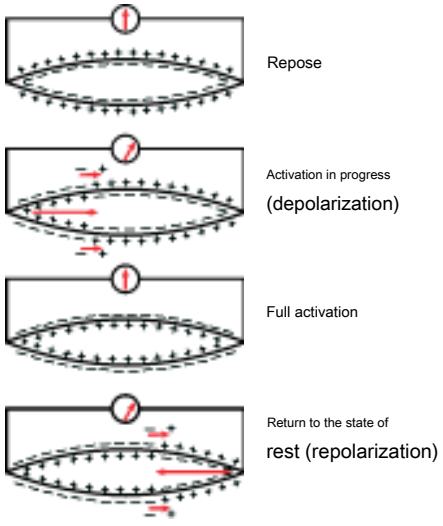
In this chapter we will only discuss the most important aspects of the electrophysiological foundations of the electrocardiogram, and we will make a brief study of the normal electrocardiogram in each of its parts.

The muscle fiber at rest is polarized, which means that the external surface of the cell membrane is electropositive and the internal surface is electronegative. The excitation of this muscle fiber produces a depolarization, that is, more precisely, an inversion of polarization: the external surface of the cell membrane becomes electronegative and the internal surface electropositive.

The activity or depolarization is schematized as the progression of "dipoles" with a positive head and negative tail. Conversely, the return to the resting state, 1repolarization, can be schematized as the progression of “dipoles with a negative head and a positive tail, that is, the progression in the opposite direction (fig. 43.1).

When we talk about electric currents, we are indicating the movement of electrically charged particles through a medium.

Fig. 43.1 Depolarization and repolarization.



driver. In the heart and other biological systems, these particles are ions, that is, atoms or groups of atoms that have electrical charges: cations, positive electricity; anions, negative electricity. These ions move out or into the cell membrane, into an adjacent conductive medium, considered an electrolyte solution.

We can then consider that if electric current is produced during ventricular and atrial depolarization and repolarization, it is obvious that this is due to ionic movements, fundamentally migrations of Na' and K cations.

At each moment of the cardiac revolution there is, due to the large number of muscle fibers, also a large number of dipoles, which can be expressed in vector terms. All of these vectors can be added into a single instantaneous resulting vector.

These resulting vectors continue to be produced at successive moments of ventricular activation, and therefore have a common origin. If the ends of all these instantaneous vectors are joined, the vectocardiogram is obtained.

From all these approaches, we could deduce, taking into account that the heart is a three-dimensional organ, of significant volume, mass and weight, that the electromotive forces generated during depolarization and repolarization are powerful enough so that they can be detected and even measured on the surface of the body, as is done in practice, and not only on the heart. In reality, what we record are not the electrical forces themselves, as they are of small intensity, barely overflowing the periphery of the heart, but as has been well pointed out, behind every electrical current there is a pressure called electrical potential, which is revealed on the surface of the thorax, truly exploring the electrical field of the heart. What is actually recorded and measured are potential differences between two scanned points of an electric field, or between a scanning point and another point of zero potential.

ELECTROCARDIOGRAM

CONCEPT

For all the above, we can define the electrocardiogram as: the graphic record of the potential differences existing between different points of the electric field of the heart or between one point of the same and another whose potential remains equal to zero (central terminal of the electrocardiograph).

SEMIOGENESIS OR PHYSIOPATHOL0GY

Transmembrane action potential of a ventricular muscle fiber

How does the electrocardiographic tracing correspond with the phenomena that occur at the cellular level?

To answer this question, it is necessary to know the intracellular potential variations, in the case we studied, the potential variations of an isolated ventricular muscle fiber.

If one electrode is placed on the surface of the fiber, and another at a point of zero potential, no electrical potential is recorded: a horizontal line is obtained.

If a capillary electrode is introduced into the cell, a negative potential of approximately 95 6 90 mV (millivolt) is obtained, called the resting membrane potential or diastolic polarization potential (PRM or PD).

value that is determined mainly by the intracellular concentration of Kt, which is around thirty times greater than the extracellular concentration; that is, approximately 150

This 'normally polarized and therefore normally excitable' fiber or cell responds to electrical stimulation with transmembrane potential variations that produce a curve known as the transmembrane action potential (TAP) or monophasic action curve (Fig. 43.2).

Five phases in the curve have been described by Hoffman:

Phase 0. Loss of systolic polarization or depolarization, there is rapid entry of the Nat cation into the cell, increasing the intracellular positivity until the potential is brought to +20 mV.

Phase 1. Initial rapid period of repolarization; K* leaves the cell.

Phase 2. Slow or plateau period of repolarization;
Kt continues leaving and Nat entering the cell.

Phase 3. Rapid repolarization period; Ky continues to leave, it is difficult for Nat to enter.

Phase 4. Restoration of the resting potential; The mechanisms called "Na pump" and "K* pumps" come into action to return the initial ionic balance of the membrane. Energy, obtained through the ATP/IATPase system, is therefore needed for the Na pump.

During phases 0; 1;2 and part of 3 no stimulus can generate another curve, it is the absolute refractory period. However, in the latter part of phase 3, a stronger stimulus may elicit a response: relative refractory period; It is followed by a period of supernormal excitation, in the terminal portion of phase 3 and the beginning of phase 4, in which a weak stimulus can produce another action curve. The excitation threshold is -60 mV.

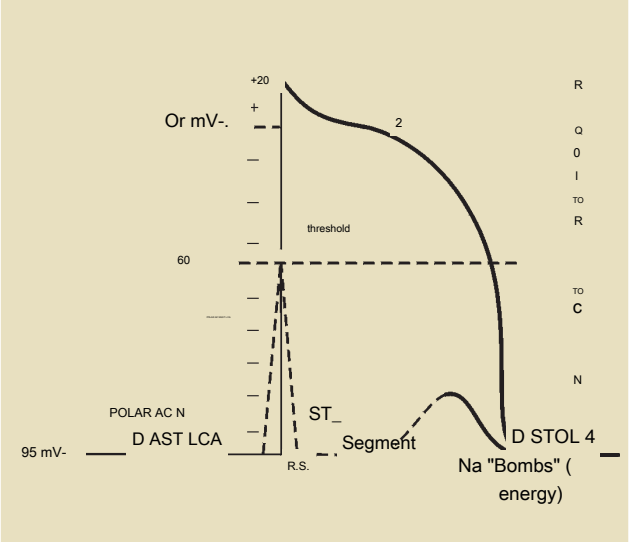


Fig. 43.2 Transmembrane action potential.

Electrocardiographic leads

We repeat that all these electrical phenomena occur in the three dimensions of space; In practice, its projection on the frontal plane and on the horizontal plane is used.

In man, the recording of potential differences is not done directly over the heart, but from a certain distance.

Einthoven's hypothesis assumes that the vectors of cardiac activation are found in the center of an equilateral triangle whose vertices would correspond to the right arm, the left arm and the left leg.

A lead is a system for exploring the cardiac electrical field. There are two types of derivations:
bipolar and unipolar. And depending on the plane in which the bioelectric phenomena are recorded, they can be derivations of limbs and precordials. Limb leads use electrodes that are placed on all four limbs and measure potential differences in a frontal plane; while the precordial leads do so in a horizontal plane and are so called because they use the electrodes located in the precordial region.

In bipolar there are two exploring electrodes, both sensitive to the potential variations that result during the cardiac activation process.

In unipolar leads, only one of the two electrodes explores the electric field, the other is connected to a point whose potential is practically equal to zero, the so-called terminal center or indifferent electrode.

Returning to the plans, we can now classify the electrocardiographic leads into:

Bipolar limb or peripheral leads

They use two of the electrodes of the four limbs.

In reality, the electrode located on the right leg is null or equal to 0, which alters the direction of the electrode placed on the left leg, we can place it further to the right, in the center of the electrodes of both legs, which corresponds to the pubic symphysis. That is to say, when we talk about the positive electrode on the left leg, its most real situation, from where it measures the potential differences, is in the pubic symphysis.

Bipolar limb leads are also known as standard leads; These are (fig. 43.3):

DI. Potential differences between the left shoulder (+) and the right shoulder ().

DII. Potential differences between the left leg or pubic symphysis (+) and the right shoulder (-).

DIII. Potential differences between the left leg or pubic symphysis (+) and the left shoulder, which behaves like (-).

As you can see, the right shoulder is always negative and the left leg or pubic symphysis is always positive.

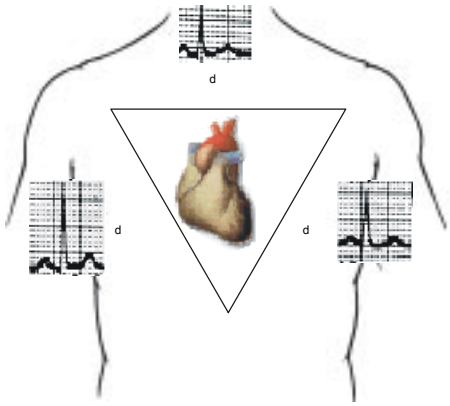


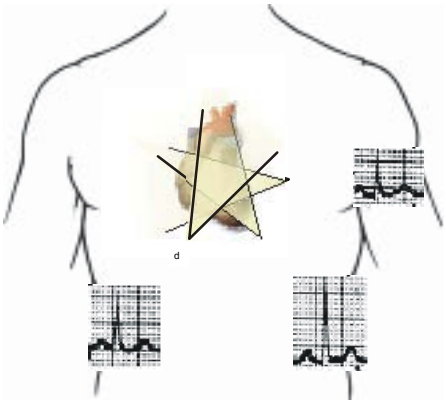
Fig. 43.3 Standard bipolar leads.

Now, since these leads measure potential differences between two poles of an electric field, the place from which cardiac bioelectric phenomena are recorded is not where the positive electrode is located in the equilateral triangle, but rather each side of this triangle, which represents an electric field, must be taken to the center of the heart, which is the center of the field; and then it turns out that the real place of each of the positive electrodes of these bipolar leads, from where the bioelectric phenomena are recorded, is the one shown in figure 43.4.

Unipolar leads

As unipolar leads record the potential differences between their positive pole and the rest of the electrodes whose potential is equal to zero, they actually record the potential that exists in the positive pole itself, without the need to transfer them to the center of the heart, like bipolar leads.

Fig. 43.4 Recording locations for leads DI, DII and DII.



a) Of the members: aVR, aVL and aVE, amplified or increased deflections. The denomination a means increased; the V, unipolar; the R, right arm; the L, left arm, and the F left leg (symphysis pubis) (fig. 43.5).

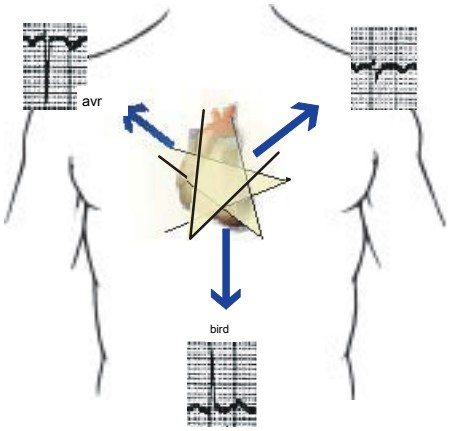


Fig. 43.5 Unipolar limb leads.

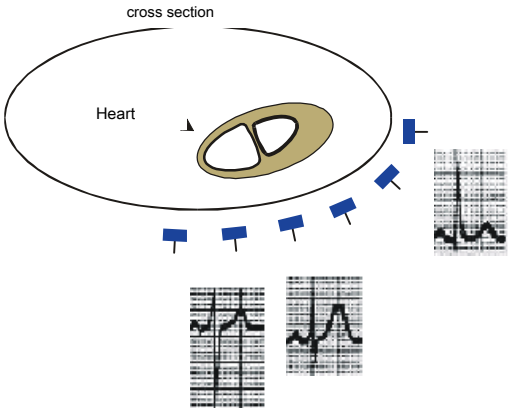
b) Precordial or thoracic: V,, V,, V,, V, V, Vo Other supplementary ones are also recorded, as we will see when we study the normal electrocardiogram (fig. 43.6) .

We insist that the leads, which register the projection on the frontal plane, are: DI, DII, DI, aVR, aVL and aVE. Derivations V, -V, record the projection of electrical phenomena on the horizontal plane.

normal electrocardiogram

Electrocardiography is a fairly precise cardiac examination method. However, its diagnostic value is not the same in all cases and it is important to know what the electrocardiogram shows in cardiovascular conditions and what it cannot show. Any alteration in the form or direction of

Fig. 43.6 The six precordial leads.



The accidents of the electrocardiographic tracing correspond to a pathophysiological state of the heart. Therefore, knowledge of the normal characters of these waves is essential. However, all cardiac conditions are not manifested by specific alterations of the normal stroke. Thus, for example, the electrocardiogram says nothing about the functional state of the heart and little about its state of compensation or decompensation. It does not give direct indications about the integrity of the coronary system itself, but only about the state of the myocardium to which it supplies.

Being an electrical examination method, it does not provide reliable information about the cause of the heart condition being explored, nor does it always provide definitive elements regarding the prognosis of heart disease. In this case, as in all examination methods used in clinical practice, the evaluation of the patient is the responsibility of the doctor, who must do so after he or she has synthesized all the elements collected and evaluated this in a complete examination.

However, cardiology has taken a giant step since the introduction of the electrocardiograph in the systematic study of the heart. Its value in the study of arrhythmias, blockages, hypertrophies, congenital diseases, and above all, ischemic heart disease, justifies its knowledge not only by cardiologists, but by every general practitioner.

Electrocardiograph

The heart produces electrical current with each beat, which is transmitted to the surface of the body through the tissues. It is collected from the body surface by electrodes and conveyed to the electroreceiving device called the electrocardiograph, which allows the aforementioned current to be recorded graphically on paper. In clinical practice, the most used ones have the galvanometer as an essential element, although they differ from the original because they are varieties of the Duddell oscillograph.

Electrocardiographs can be single-channel or multi-channel, depending on whether they record a single lead or several at the same time; In the latter case, it allows the simultaneous analysis of cardiac activity in different leads.

Whatever the type of device, the electrocardiogram records the graph of cardiac current variations as a function of time.

When no electric current circulates in the device, it inscribes a horizontal line, called isoelectric line;
Current variations determine positive or negative deflections that make up the complexes called the atrial complex (P) and the ventricular complex (QRS-T).

It is understood that the dimension of a deflection depends on two factors:

1. The voltage of the electric discharge (expressed in millivolts, above or below the isoelectric line).
2. The duration of said deflection (expressed in hundredths of a second).

The dimension of each deflection can be obtained very precisely, considering the surface covered by said deflection on the isoelectric line.

In order to compare traces, it is internationally accepted to adjust the sensitivity of the devices in such a way that one millivolt (1 mV) determines on the paper a deflection of 1 cm (10 mm) in all the leads, although some prefer half of said sensitivity ½mV=0.5 cm for the precordial leads, especially when the deflections are greater than the paper and the fountain pen collides at the ends, deforming the tracing.

On the other hand, the equipment is designed so that the paper moves at a standard speed of 25 mm/s, although many equipment also has a speed greater than 50 mm/s. Since the paper is squared at I mm, the standard speed of the equipment is 25 small squares/s, which is equal to 1,500 squares in l min. If 25 squares pass in one second, the average time it takes for each millimeter or square to pass is 0.04 s (1/25).

SEMIOTECHNICS

We have already seen that in standard or peripheral leads potential differences between two extremities are explored, placing the exploring electrodes on the right arm, the left arm and the left leg; that the unipolar limb leads are those that register the potential variations, in each limb separately, and that the precordial leads are those that register the potential variations in the precordial area, by an exploring electrode at the following six points:

V, Right border of the sternum, at the level of the fourth intercostal

V, Left edge of the sternum, at the level of the fourth intercostal

V, Point located in the middle of the line that joins V, - V,.

V, Point located at the intersection of the left midclavicular line with the fifth intercostal space.

V, Intersection of the left anterior axillary line with a horizontal line that passes through V

V, Intersection of the left midaxillary line with a horizontal line that passes through V4'

In some cases it is necessary to explore points located further to the left of V,: they are the points V, ,V,and V,; or further to the right of V,: they are points 2r and 3r, or point E, located on the xiphoid process.

SEMIOGRAPHY OF ITS PARTS

The normal human electrocardiogram graph is made up of a series of waves, segments and intervals.
Waves since Einthoven have been designated by the letters P,Q, R, S, T, and sometimes U (fig. 43.7).

P wave

It is the record of the atrial depolarization that precedes and corresponds to the simultaneous contraction of both atria. It is rounded and generally positive, 2.5 mm by 2.5 mm; That is, its average duration is 0.08-0.10 s. Its amplitude varies from 1 mm and should not reach 3 mm.

SegmentP or PR

It records the time it takes for conduction, normally through the A-V node, on its way to the ventricles. It is the isoelectric line segment between the P wave and the QRS complex. It normally measures around 2 mm (0.08 s).

PR or P interval

It is the space of time between the beginning of the P wave and the beginning of the QRS complex (beginning of the Q wave, when it exists, or the beginning of the R wave, when the Q wave does not exist). It includes the P wave and the PQ segment.

It corresponds to the pause that extends from the beginning of atrial excitation to the beginning of ventricular excitation. It also corresponds to the time of atrioventricular conduction. Its duration normally varies with heart rate and age; It is shorter in tachycardia and in children. Its duration (normal value) varies around 0.16-0.20 s.

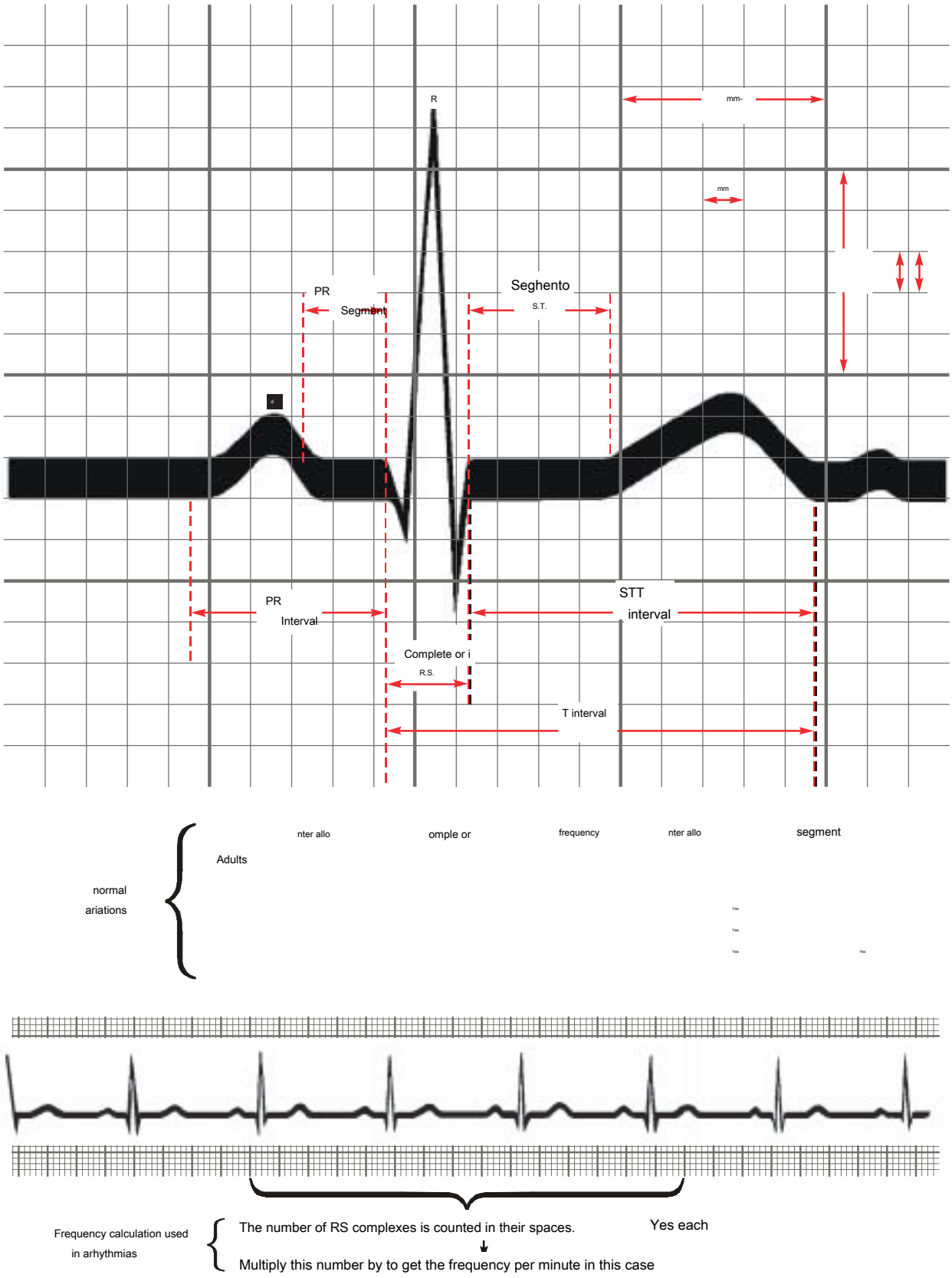


Fig. 43.7 Waves, segments and intervals of the electrocardiogram and one of the ways to find the heart rate.

RS Complex

It results from ventricular activation or depolarization and precedes the contraction of the two ventricles, with which it is associated. The main feature of this normally tall, thin, positive complex is the one designated by the letter R. This is the first positive wave of the complex, which may or may not be preceded by a small, negative wave. and is then followed or not, by another negative wave, S.

The QRS complex normally lasts up to 0.08 s in adults and around 0.06 in children. If it is 0.12 s or more, it is suggestive of a disorder in intraventricular conduction known as complete bundle branch block. Its amplitude (height) varies depending on the electrical position of the heart in the tracing, the degree of ventricular growth, and other factors. Its average value in the peripheral leads is 10 mm =1 mV.

ST segment

It extends from the end of the S wave (or the R deflection, when S does not exist) to the beginning of the T wave. It corresponds to the period of sustained contraction of the ventricles. Nacey is normally inscribed on the isoelectric line. An inscription of more than 1 mm above or below this line is associated with an abnormal state (infarction, ischemia or vagotonia).

T wave

With the ST segment, the T wave is of great importance in electrocardiography. The ST-T set constitutes the terminal phase of the ventriculogram.

The T wave corresponds to the recording of the return to the resting state of the ventricles; that is, repolarization and ventricular electrical excitability. It is normally registered as an asymmetric wave, with a slow initial branch and a fast final branch; It is generally positive, 5 mm by 5 mm on average; that is, 2-6 mm in amplitude and 0.20 s in duration.

More important is to know that the T wave can normally be up to a third of the height of the corresponding R wave. For example, if an R wave is 21 mm, the T wave can be up to 7 mm.

T interval

It extends from the beginning of the QRS complex to the end of the T wave and measures the duration of ventricular systole quite accurately. Its normal value is 0.36 ±0.04 s for a heart rate of 70 contractions per minute. Its duration is prolonged in heart attacks, ischemia, hypocalcemia, hypoparathyroidism, tetany, rickets, etc. It is shortened in hypercalcemia and with the use of digitalis.

U wave

It is fickle; It resembles a shorter T wave with lower amplitude. It is frequently observed in bradycardia, vagotonia and in the hearts of athletes. Also in high blood pressure and diabetic coma.

METHOD OF READING AN ELECTROCARDIOGRAM

Reading an electrocardiogram is not achieved without a systematic analysis of each of its accidents; For this it is good to always proceed in the same way and according to a pre-established plan.

Several methods have been proposed for the systematic reading of the electrocardiogram. It is very useful to follow the following steps:

Determination of heart rate (pacemaker).

Determination of heart rate.

- Electric axle.

- Electrical position.

Study of the P wave.

Study of the PR interval.

- Study of the QRS complex, the Q wave, the R wave and the S wave.

Study of the ST segment.

Study of the T wave.

QT interval study.

Study of TP space.

At the end, a summary and interpretation of the analyzed alterations must be made.

To read an electrocardiogram, it is useful to have a two-point compass in order to measure the regularity of the spaces and waves. It is also useful to have graphs on hand that give the value of some spaces as a function of heart rate.

Before interpreting the tracing, you must ensure that the designation of the leads has been done correctly and that there are no artifacts or interferences due to: poor contact of the electrodes; incorrect electrode placement: patient conditions, such as tremor, muscle tension, anxiety, little or no relaxation:

nearby electrical equipment, etc.

We will also ensure that the standardization of the layout is correct: 1 mV= 10 mm in all leads. If ½ mV=5 mm is used for the precordial leads, it should be noted on the tracing.

Furthermore, before forming any concept about the layout, it is prudent, to avoid making gross errors, to make sure:

a) The age of the patient.

b) From the position in which the drawing was taken (sitting, lying down).

c) Of the medications that the patient has been taking (digital, others).

d) The probable clinical diagnosis.

We will now describe the steps indicated in reading an electrocardiogram.

Determination of pacemaker heart rate

We are interested in pointing out here the electrocardiographic criteria to be able to diagnose the normal rhythm of the heart, the regular sinus rhythm; Later we will study rhythm alterations, arrhythmias.

To determine the normal rhythm of the heart or regular sinus rhythm, the:

a) The morphology of the P wave in two unipolar limb leads: aVR and aVE. If there is no P wave, we think of a rhythm originating from the ventricles themselves. If there is a P wave and it is positive in aVF and negative in aVR, the pacemaker is located in the sinus node or in the atrial heart muscle. Otherwise, we think of retrograde activation of the atria, from bottom to top, therefore, in a rhythm originating in the lower portion of the atrium or in the upper portion of the atrioventricular junction.

b) The regularity of the rhythm that is required by measuring several P-Po R-R spaces, that is, the time elapsed between the beginning of two successive P waves or the peak of two successive R waves, that is, a cardiac cycle. In a regular rhythm the chosen interval does not vary by more than 0.12 s. Otherwise it is called arrhythmia. The P-P and R-R intervals must be equal in the same lead and throughout the ECG each P must precede a QRS.

c) The atrioventricular conduction time, which must have several PQ segments and successive PR intervals, of normal and constant duration.

d) The morphology of P, which must be the same in the same lead, with the exception of leads DII and aVF in which there may be changes induced by respiration.

In summary, ordinary or regular (or normal) sinus rhythm has the following characteristics:

a) P-P or R-R must measure more than 15 mm and less than 25 mm in width; that is, between 0.60 and 1 s (average: 0.85 s for a frequency of 70 beats/min) exhibiting variations of less than 0.12 s.

b) Frequency of 60-100 beats/min.

c) Equal PQ segments and constant and normal PR intervals, from 0.12 s to 0.20 s.

Determination of heart rate

Some tables give the frequency in relation to the length of an R-R interval.

As we already said, according to the speed of the equipment, the space between two consecutive thin vertical lines on the electrocardiographic paper that delimits I mm is equivalent to 0.04 s. Each space of five thin lines (marked on the paper by a stronger line, which delimits the width of 1 large square) is equal to 0.20 s. Elo means that the paper moves at a rate of 1,500 small squares (millimeters) or 300 large squares in one minute.

The classic method for finding the frequency is to divide 1500 by the number of squares separating two R waves in a lead. Example: if there are twenty squares between two R's, 1, 500/20 = 75 beats/min.

For a quick calculation, you can use the following resource; Subtract from 155 the hundredths of a second that separate two R waves. For example: If R-R = 0.80 s, the frequency will be: 155 – 80 = 75 beats/min.

The fastest and most practical calculation in our opinion is to divide 300 by the number of large squares that separate two R waves in a lead. So if two R's are separated by 1 large square, the rate is 300 beats/min; 2 large paintings, 150; 3 frames, 100; 4 paintings, 75; 5 frames, 60; and 6 large paintings, 50/min. This method can only be used at regular frequencies; that is, with equal R-R intervals.

Another practical method to determine the frequency is obtained by counting how many R-R intervals fit into 15 cm of the tracing, 30 large squares (6 s), and multiplying this figure by 10, the result of which corresponds, quite precisely, to the heart rate. It can also be obtained by counting the QRS that contain 15 large squares (3 s) and multiplying it by 20. It is the method used to determine the heart rate in arrhythmias, when there is irregularity in the R-R intervals (see Fig. 43.7).

electric shaft

In clinical electrocardiography it is useful to determine and quantify to the extent possible the frontal and anteroposterior directions of P, QRS and T, using for the frontal plane the Einthoven triangle (Fig. 43.8) and/or the three-axis (triaxial) or six-axis (Bailey and Cabrera hexaxial) reference systems (Fig. 43.9). The frontal projections of the spatial mean axes are called manifest mean axes of P, QRS and T, simplifying, we speak of P, QRS and T axes.

Of these last three axes, the most important is the manifest middle axis of QRS or ventricular electrical axis, because it determines the main direction of the ventricular loop of the vectocardiogram, and it will be the one where we will stop in its determination. The manifest middle QRS axis is commonly called the electrical axis of the heart, and is identified as AQRS.

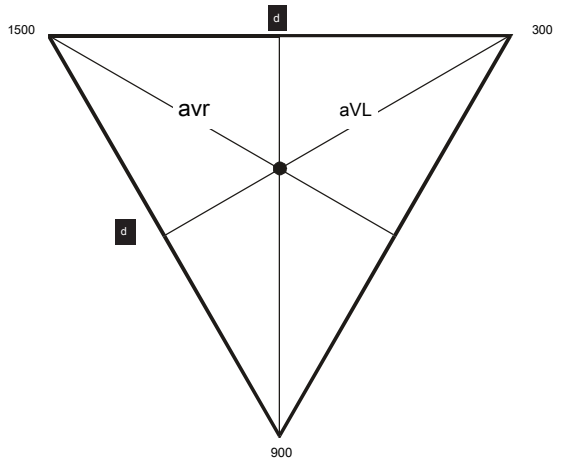
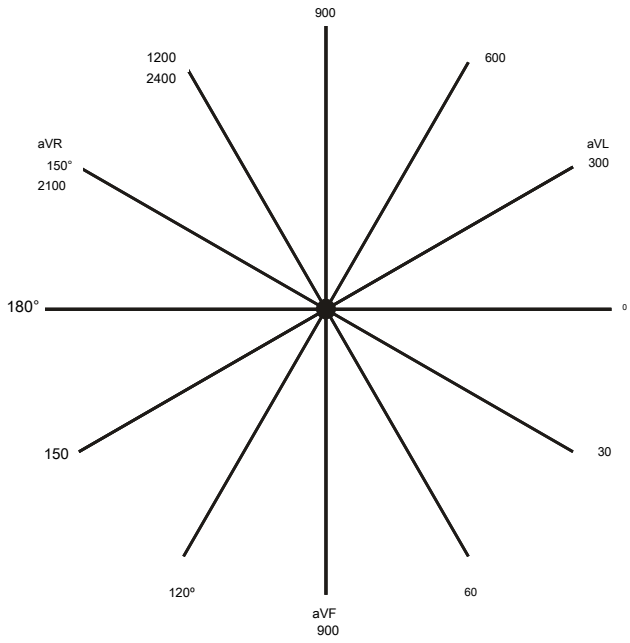


Fig. 43.8 Einthoven's triangle.

Fig. 43.9 Hexaxial system.



Classic method to find the ventricular electrical axis

To determine the electrical axis of QRS, the classic way is that used by the Bailey triaxial system.

This three-axis system is built from the Einthoven triangle, moving the sides of the triangle and preserving its original direction until the midpoints of these three sides coincide with an imaginary point placed in the center of the triangle.

We divide each of these three axes thus constituted by an arbitrary measure chosen, which represents each unit. If we draw an imaginary circle around this triaxial system, choosing as the center the imaginary point placed in the center of the triangle, it is automatically divided by these three axes in six portions or sextants. The top three are negative and the bottom three are positive.

To determine the electrical axis we use the lines of leads DI and DIII, and proceed as follows (fig. 43.10):

a) The algebraic sum of the largest positive wave with the largest negative wave in lead DI is performed and the value found is taken to the line of lead DI in the system, taking care to mark this value on the corresponding side (positive or negative).

b) The same procedure is followed in derivation DIII, determining another point on the line of derivation DIII.

c) At these points we raise perpendicular to the line of the corresponding derivation. These two perpendiculars will intersect at a

d) Using a line, the point where these perpendiculars have been cut is joined to the center of the triangle, thus forming the body of the vector, whose head is placed at the point where the perpendiculars were cut, where an arrow is drawn.

The normal electrical axis is very variable, especially with age, it is generally between 0° and + 90°. However, when the axis approaches 0° there is a tendency towards left axial deviation, if it approaches + 90° there is a tendency towards right axial deviation.

Other methods to find the electric axis

Before describing them, it is necessary to make some important considerations.

The ECG equipment is prepared so that, when the depolarization wave moves towards the positive pole, the needle deflects upward, called positive; and when it moves away from the positive pole it inscribes a downward deflection, called negative.

The QRS complex shows a deflection that varies according to the direction in which the ventricular depolarization wave travels (Fig. 43.11).

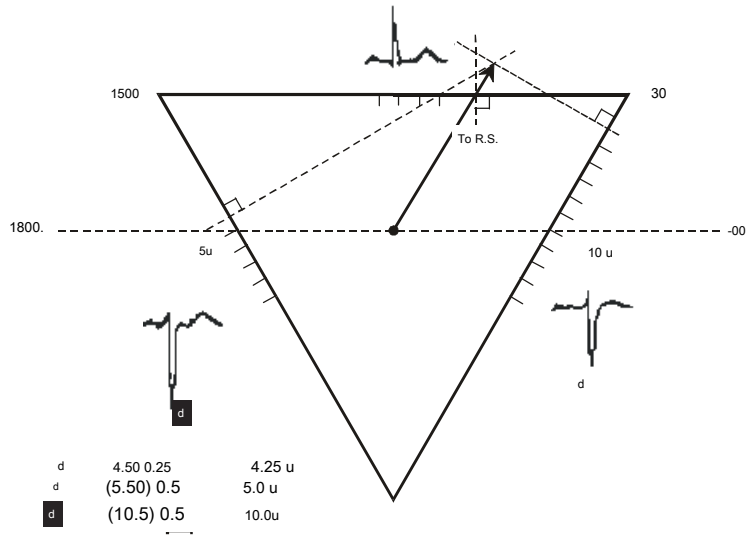
If ventricular depolarization shifts toward the positive pole, the QRS complex will be predominantly upward or positive. That is, the R will be greater than the S (Rs).

If ventricular depolarization moves away from the positive electrode, the QRS will be predominantly downward or negative. That is, the S will be greater than the R (rS).

If the ventricular depolarization moves perpendicular to the positive electrode, then the size of the R and S will be equal (RSors). That is, the QRS will be made up of two equal phases, one positive and one negative. The QRS will be isophasic.

The Q wave has a different meaning, which will be studied later.

Fig. 43.10 Determination of the electrical axis by the classical method.



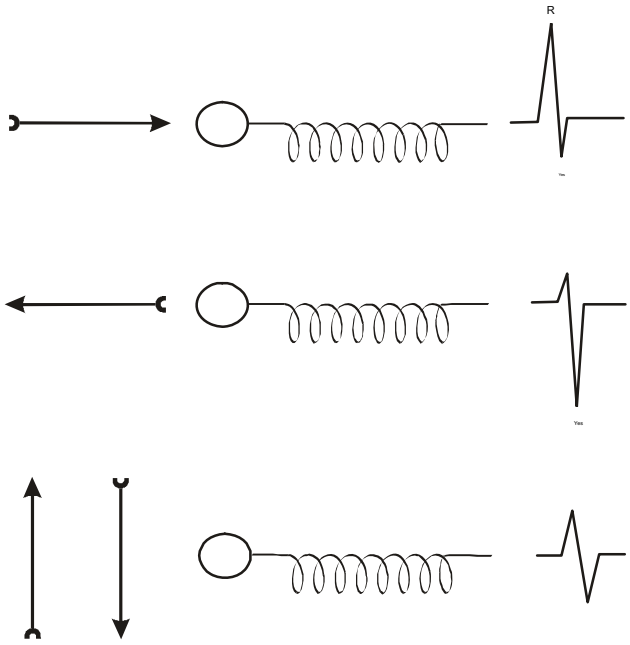


Fig. 43.11 Shape of the RS according to the direction of ventricular depolarization in relation to the positive pole of the lead.

On the other hand, the ventricular depolarization wave propagates in different directions and gives rise to many vectors, of different magnitudes and directions. If we want to know the magnitude, direction and final sense of the displacement of the depolarization wave, we must geometrically add all the vectors and we will obtain a resulting vector.

Normally, the vector resulting from the depolarization wave of the heart, seen from the front, propagates from top to bottom and from right to left.

If we compare its direction with the time on a clock face, we would say that it spreads from 11:00 to 5:00 (fig. 43.12).

So we can also use as a definition that:

The resulting direction of the depolarization wave seen from the front is called the electric axis.

Determining the electrical QRS or ventricular axis is important as an indirect sign of ventricular growth. This may be normal, deviated to the left, deviated to the right, or in the indeterminate quadrant.

If we take the heart as the center of a circle and of a coordinate axis simultaneously,

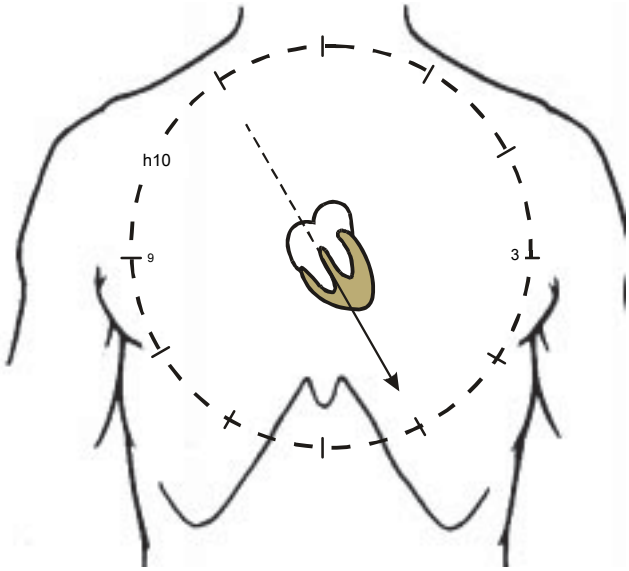


Fig. 43.12 Direction and meaning of the result of normal depolarization.

te, the circumference is divided into four quadrants (fig. 43. 13).

As can now be seen, in figure 43.14, it has been established in a conventional way that the right horizontal line is the initial point, which marks 0°. Following the clockwise direction, positive values are given to the different angles; and counterclockwise, negative values are given. Since the upper left quadrant can have positive and negative values, depending on the direction taken, it is called the indeterminate quadrant.

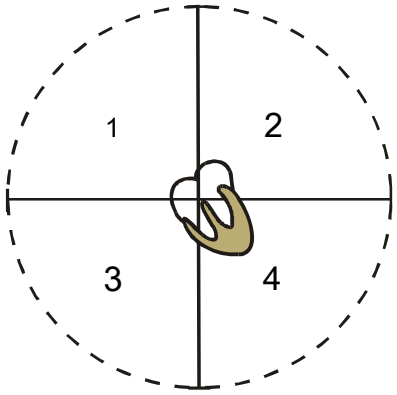


Fig. 43.13 The four quadrants of the circumference, whose center is the heart.

The normal electrical axis is the one between 0° and +90°. When the direction of the electric axis is between O° and -90°, there is left axial deviation and when it is between + 90° and + 180° there is right axial deviation.

If the electrical axis is between - 90° and + 180° we say that the electrical axis is in the indeterminate quadrant.

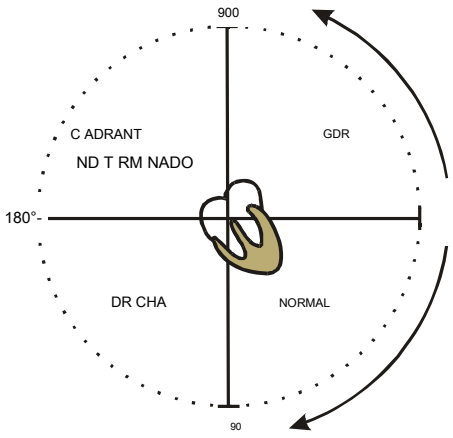


Fig. 43.14 Names and degrees of the quadrants.

The electrical axis in the indeterminate quadrant can be seen in some rare congenital heart diseases and it is almost always an expression of ventricular growth extreme right.

Remember, the electrical axis is found with the limb leads.

Method using leads D,D and D

By this method, the ventricular electrical axis is found from the shape of the QRS in the three bipolar limb leads, and of these, the most important are DI and DII.

In accordance with the principles that we have already expressed in this chapter, related to the direction of the inscription: positive, negative or isophasic, depending on whether the depolarization approaches, moves away from or is perpendicular to the positive electrode, respectively, it is considered that the depolarization approaches the positive pole of each lead, when it is located in the area between a perpendicular drawn at the midpoint and the positive pole (fig. 43.15).

Let's now see what happens in bipolar limb leads, when the electrical axis is normal, is deviated to the left, is deviated to the right, and when it is in the indeterminate quadrant.

If the electrical axis is normal, the depolarization wave approaches DI, DII and DIII and, therefore, the QRS of these three leads will be predominantly positive.

Note that when the electrical axis is around + 60° it is closer to DII than to DI and DIII and that is why the QRS is more positive in the former than in the other two (fig. 43.16).

If there is left ventricular growth, or for any other reason, the electrical axis deviates to the left, the direction of the depolarization wave approaches DI and moves away from DIII. That is, DI will be predominantly positive and DIII, predominantly negative.

When the electrical axis is deviated to the left, the QRS complex in DII is almost never predominantly positive; It will be isophasic or with a negative predominance (Fig. 43.17).

If, for example, there is right ventricular growth and right axial deviation occurs, the depolarization wave moves away from DI and approaches DIII. Therefore, DI will be predominantly negative and DIII, predominantly positive (Fig. 43.18).

If the electrical axis is in the indeterminate quadrant, the depolarization wave moves away from DI, DII and DIII. The QRS complex in these three leads will be predominantly negative (Fig. 43.19).

If we now analyze the topic in the opposite direction, from the electrocardiographic tracing to the determination of the electrical axis, we have the possibilities summarized in Figure 43.20.

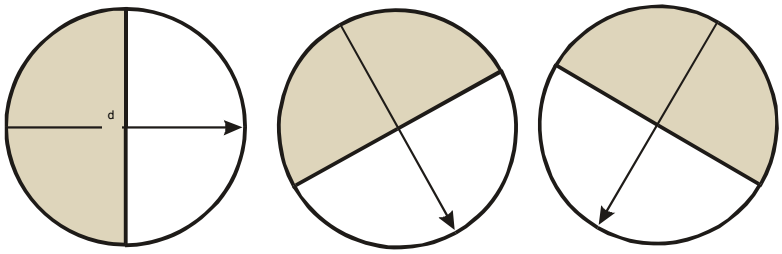


Fig. 43.15 Depolarizations approach the positive electrode if their resulting vector is in the light zone and move away if their vector is in the clear zone.

DARK.

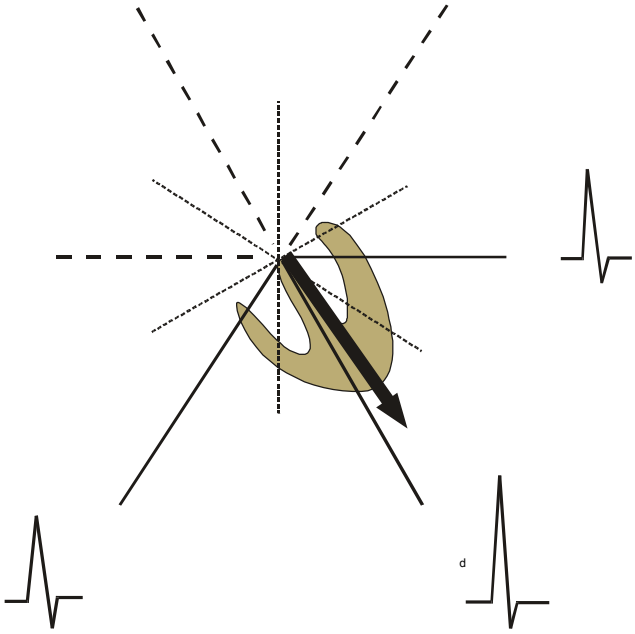


Fig. 43.16 Shape of the RS in DI, DII and DIII with a normal electric axle.

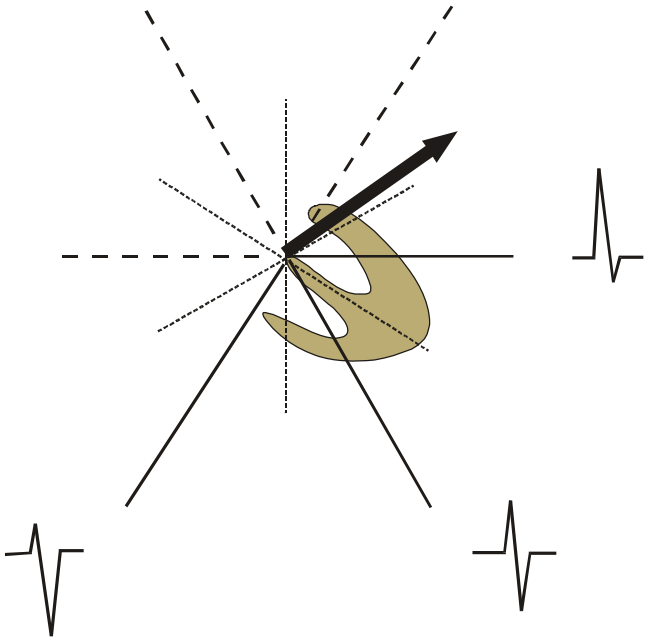


Fig. 43.17 Shape of the RS of DI, DII and DII in the left axial deviation.

Fig. 43.18 Shape of the RS of DI, DII and DIII in the right axial deviation.

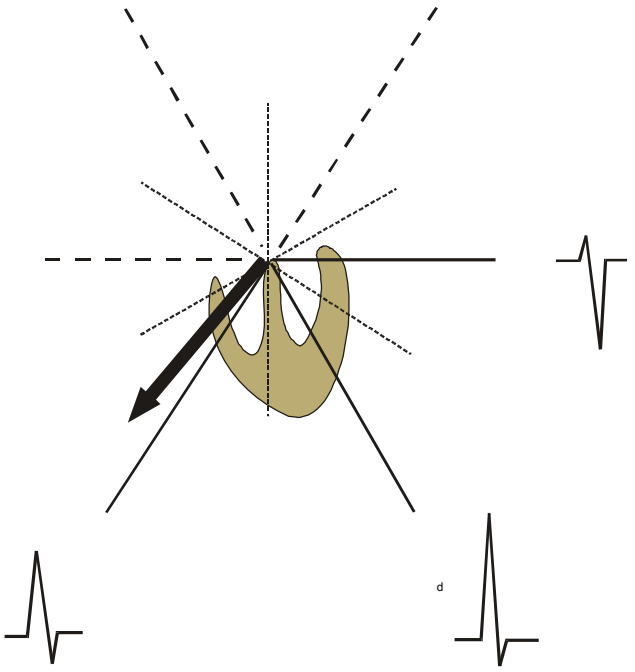


Fig. 43.19 Shape of the RS in DI, DII and DII with the ventricular axis in the indeterminate quadrant.

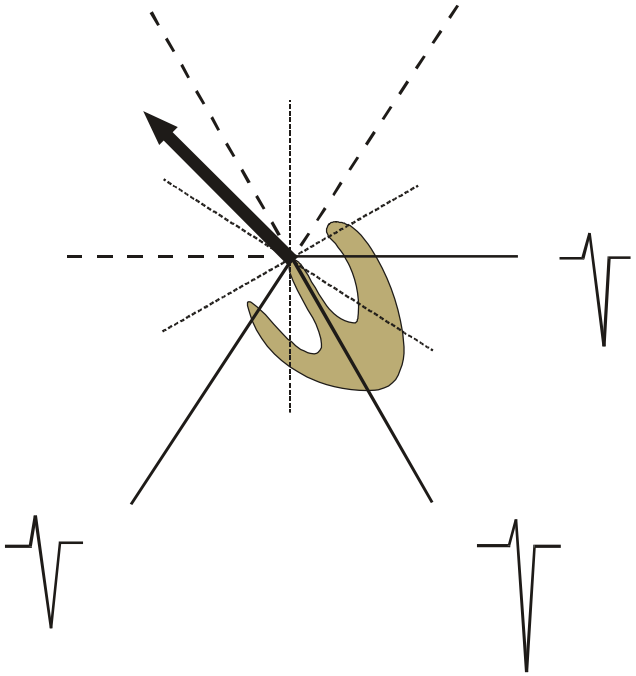
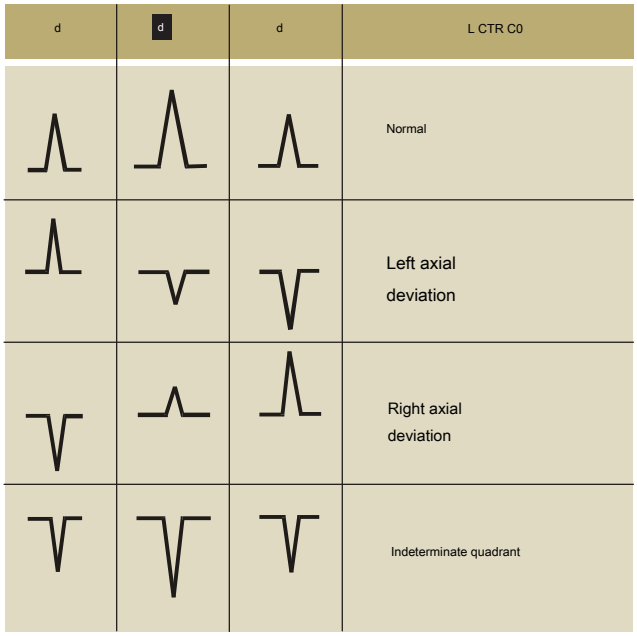


Fig. 43.20 Ventricular electrical axis according to RS shape in DI, DII and DII.



Method using leads D and aVF

There is another method to find the electrical axis, according to the shape of the QRS in DI and aVE.

If DI is predominantly positive, the electrical axis may be normal or deviated to the left and if it is predominantly negative it will be deviated to the right or in the indeterminate quadrant (fig. 43.21).

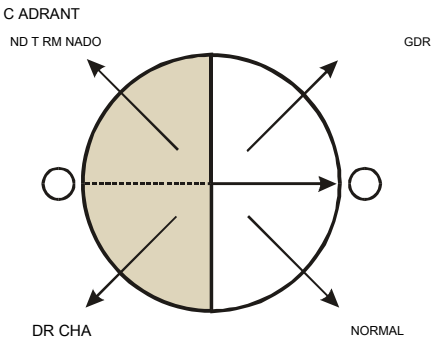


Fig. 43.21 Possible situations of the electrical axis, according to its positive or negative predominance in DI.

If aVF is positive, the electrical axis may be normal or deviated to the right, but if it is predominantly negative it will be deviated to the left or in the indeterminate quadrant (fig. 43.22).

The electrical axis will be where DI and aVE coincide.

Summing up (fig. 43.23):

DI (+) and aVF (+) ..... Electrical axis

DI (+) and aVF () ..... Electrical axis to the i ie d

DI () and aVF (+) ..... Electrical axis to that of ec DI (and aVF (.... Axis in indeterminate quadrant

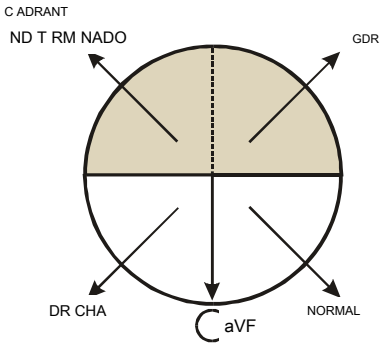
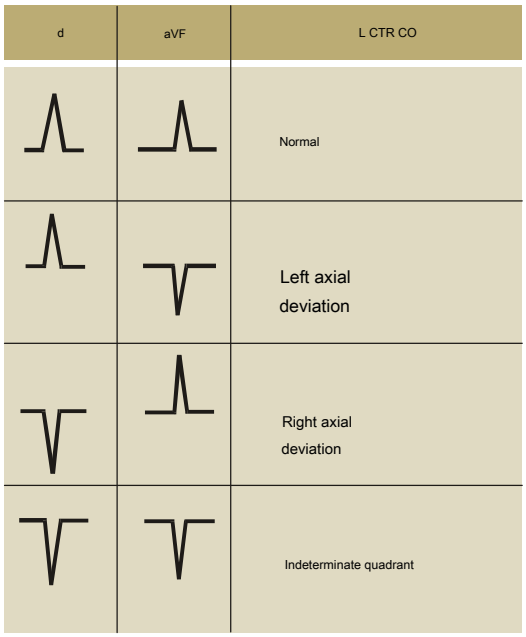


Fig. 43.22 Possible situations of the electrical axis, according to its positive or negative predominance in aVF.

Fig. 43.23 Ventricular electrical axis according to RS shape in DI and aVF.



This last method is the most suitable for finding the electrical axis quickly and without errors. But if you want to find more accurately the degrees of the direction of the electrical axis, see more details on this topic in the electrocardiography texts.

electrical position

The interpretation and meaning of electrical position have been one of the most controversial topics in electrocardiography.

In the light of vectorcardiography, the electrical position, as well as the electrical axis, are indicators of the mean orientation of the vector resulting from the ventricular depolarization process in the frontal plane. The rotation of the heart on its different anatomical axes, al-

tera the position of the middle electrical axis of the QRS complex in the frontal plane. Rotation around the anteroposterior axis occupies a prominent place in this sense, as well as that which almost always occurs concomitantly about the longitudinal axis of the heart.

Classically six electrical positions are described:

1. Horizontal.
2. Semihorizontal.
3. Intermediate.
4. Semi-vertical.
5. Vertical.
6. Indeterminate.

The determination of the electrical position is carried out by studying the unipolar leads of the aVL and aVE limb, which show the electrical potentials of the cardiac source from practically opposite points, therefore offering an ideal situation for the analysis of the orientation of the vector in the frontal plane.

Electrocardiographic criteria:

1. Horizontal position (axis at -30° or beyond). In lead aVL it will exhibit a strong positive predominance of QRS, while in aVF it will be negative: R in aVL and S in aVE.

2. Semi-horizontal position (approximately 0°). Strong positive predominance in aVL with isophasic or small voltage QRS in aVF: R in aVL and rs in aVF.
3. Intermediate position (+ 30°, approximately). Positivity predominates in both leads: R in aVL and aVE

4. Semi-vertical position (+ 60°, approximately). Strong positive predominance in aVF with isophasic QRS equal to zero in aVL: R in aVF and rs in aVL.
5. Vertical position (axis at +90° or beyond). Predominance of positivity in lead aVF and aVL to strong negative predominance: R in aVF and S in aVL.
6. Indeterminate position (axis between -120° and +180). In both leads there is a negative predominance of the QRS complex: S in aVL and aVF.

Study of the P wave

The P wave is the first accident of the electrocardiogram. Translates atrial depolarization. Normally

Its duration is 0.08 s, it is considered normal up to),11 s.

Its amplitude varies from 1-3 mm. It has a rounded shape, slow inscription and thick lines.

It is generally positive in the three standard leads, negative in the aVR lead. Its greatest positivity is found in DII, and in DIII it can be flattened, isoelectric or even inverted without necessarily being abnormal (fig. 43.24).

Its measurement is best performed in lead DII.

PR interval study

It is the sum of the P wave plus the PQ or PR segment (from the termination of the P wave to the beginning of the QRS complex) , its measurement is essential and has great value.
Its normal duration varies between 0.12-0.20 s, depending on the patient's age and heart rate. It is generally measured in DII where the interval is the longest.

Study of the RS complex, the R wave and the S wave

In the tracing, the QRS complex represents ventricular depolarization (septum and both ventricles). Although it is called the QRS complex, these three waves do not necessarily have to appear, and the complex presents itself with numerous variants. Its waves are identified as follows:

a) The R wave is always positive. If more than one appears, they will be called R, R', R", etc.
b) Any negative deflection that precedes the R wave is a Q wave and this is always negative when it exists.
c) Every negative wave that follows the R wave is a wave S and this is always negative.
d) When any of these waves is small, it is designated by its corresponding lowercase sign q, r or s.

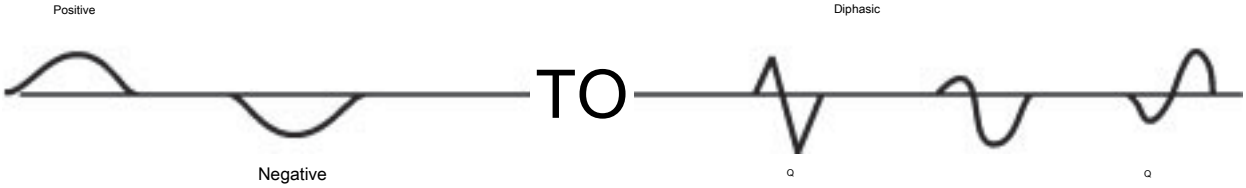
The QRS complex is considered positive or negative when the algebraic sum of the areas inscribed by each wave gives a positive or negative result.

When the R wave does not exist, the QRS complex is composed only of a negative deflection, called QS.

The normal QRS complex lasts between 0.08 and 0.10 s.

The study of the characteristics of the Q wave when it is present is essential. It is accepted that in the normal adult a Q wave should not exceed 25% of the R value in each lead. In aVR the Q wave is normally pro-

Fig. 43.24 P wave morphologies.



sheath. In right precordial leads there are normally no Q waves (V, V, and V). In V,, V, and V, there may be a small Q wave, proportional to the height of the R wave, but its presence must be carefully studied.

The OR interval (duration of intrinsicoid deflection) extends from the beginning of the Q wave when it exists, or of the R wave itself, to its apex. Its maximum value is 0.035 s in the right precordial leads and 0.045 s in the left.

Study of the ST segment

It is a thick, isoelectric trace that goes from the end of the QRS complex to the beginning of the T wave; represents the absolute refractory period of the heart. It separates the process of ventricular depolarization (QRS) from that of repolarization (T wave).

T wave study

It is a slow, rounded, thick wave that follows the ST segment and represents the process of ventricular repolarization. It is asymmetrical, since its proximal branch must have a much slower inscription than the distal one. A symmetrical T wave is generally pathological, especially if it is pointed.

The T wave is normally positive in DI, DII, DIII, aVL and aVE. It is normally negative in aVR.

In left precordial leads, the T wave is normally positive; On the other hand, on the right side of the precordial area, the T wave is usually negative in V,.
Occasionally, T wave negativity may be normal up to V, in young people, blacks, and women.

beef.

Study of the T interval

It is the time interval that goes from the beginning of the Q wave to the end of the T wave, including, therefore, the sum of the QRS complex, the ST segment and the T wave. It represents the process of ventricular activity (depolarization + repolarization). Its normal value for a frequency of 70 beats/min is 0.36 + 0.04 s.

TP space study

It is the thick, isoelectric trace that appears after the T wave. It is measured from the end of this wave to the beginning of the P wave of the following complex. It is of great interest in the study of arrhythmias. If there is a U wave, its morphology is noted.

The PT space corresponds to ventricular diastole. Sometimes the U wave appears over this space, in which case it is usually of low amplitude. Its significance is not well determined; It can occur in cases of hypokalemia, vagotonia, in athletes' hearts and in hypertension.

ELECTROCARDIOGRAPHIC ALTERATIONS

FUNDAMENTALS

44

Introduction

Having discussed the normal electrocardiogram in the previous chapter, we now move on to the study of the fundamental electrocardiographic alterations:

1. P wave alterations:
   - Atrial enlargement or hypertrophy.
2. QRS alterations:
   - a) In width.
     - Branch blocks and fascicular blocks (hemiblocks).
   - b) In height.
     - Ventricular growths.
     - Microvoltage.
   - c) Presence of pathological Q.
     - Myocardial necrosis.
     - Q for diastolic overload of the left ventricle.
3. ST and T wave alterations:
   - a) Secondary alterations in ventricular repolarization.
     - To complete branch block.
     - Ventricular systolic overloads.
   - b) Primary alterations of ventricular repolarization.
     - Due to ischemic heart disease.
   - c) Other ST-T alterations.
4. Rhythm alterations (arrhythmias).
5. ECG alterations in other common diseases.

P WAVE ALTERATIONS

ENLARGEMENTS 0 ATRIAL IPERTROPHIES

Three electrocardiographic diagnostic criteria of the P wave are studied taking into consideration:

1. Duration.
2. Voltage.
3. Morphology.

The normal duration of the P wave is up to 0.11 s, its voltage does not reach 3 mm, and in terms of its morphology, it can be a normal P wave, an abnormally wide P wave, called mitral P, and an abnormally tall P wave, called pulmonary P.

So the two pathological alterations of the P wave are: abnormally wide P wave, mitral P, and abnormally tall P wave, pulmonary P (Fig. 44.1).

P wave abnormally wide to mitral P

It means growth of the left atrium. Its name is due to the fact that it was first described in mitral stenosis, but it is also observed in other diseases that affect the left heart.

Mitral P has a duration of 0.12 s or more; its height, amplitude or voltage can reach 3 mm or more, but its duration is altered more than its height. Its typical morphology is known as bimodal, due to the two modes or humps it has at its peak.

The mitral P wave is observed in DI, DII, aVL, V, and V.
It is usually flattened in DI, aVL, V, and V, bifid or bimodal in DII, small in DIII.

It should also be studied in precordial lead V, where the P wave is diphasic (positive-negative); The first positive phase corresponds to the depolarization of the right atrium and the second negative phase to the depolarization of the left atrium, and in these cases the negative phase is very delayed; That is, the negative phase is larger than the positive phase, especially in width (P) (see Fig. 43.24).

The position of the electrical axis of the P wave, when it is considered pathological due to its duration, height and morphology, can be useful to determine which atrium is causing the anomaly. When the P wave in DI and DII lasts more than 0.11 s, is bimodal and its electrical axis is deviated to the left between 45° and -30°, then we can establish the diagnosis of left atrial enlargement.

Semidiagnosis

Left atrial growth due to any cause:

1. Mitral stenosis.
2. Mitral insufficiency of any etiology.
3. Left ventricular growth of any etiology.
4. Congenital heart diseases: ventricular septal defect (VSD) and patent ductus arteriosus.

Abnormally high pulmonary P wave

It means growth of the right atrium.

It is characterized by increased height or voltage of the P wave in leads DII, DIII and aVF; its voltage is altered more than its duration. The morphology is tall, pointed, acuminate. Clearly visible in the right precordials, when it is diphasic in V, the positive phase is greater than the negative, especially in height (P*- (see fig. 43.24).

Semidiagnosis

Right atrial growth due to any cause:

1. Acquired hypertensive pulmonary heart disease: chronic cor pulmonale and pulmonary overdistension (probable emphysema).
2. Cyanotic congenital heart diseases: pulmonary stenosis and tetralogy of Fallot.

ALTERATIONS OF THE RS IN ANC URA

BLUEOS OF A BRANCH

Concept

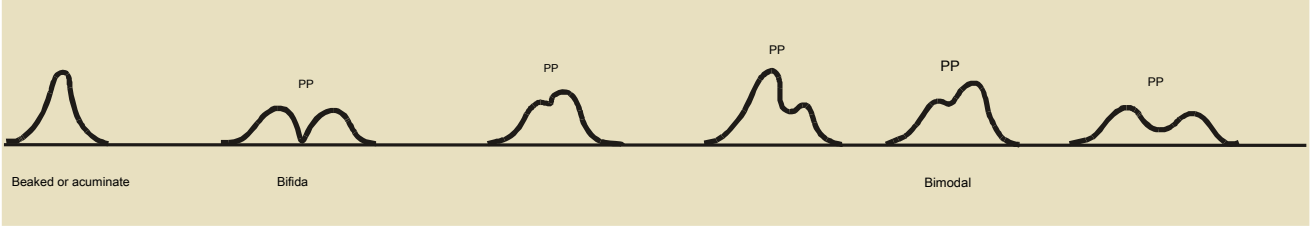
It means an interruption or delay in the passage of the electrical stimulus through one of the branches of the bundle of His.

The interruption of the stimulus is produced not only by a lesion located at the level of one of the branches, but also by sufficiently extensive lesions of the Hisian branches, of the Purkinje network or of the muscle itself.

Electrocardiographic criteria

1. Constant PR interval of a duration greater than 0.12 s.
2. Wide QRS complexes, with a duration equal to or greater than 0.12 s and with intraventricular conduction disorders (aberrant) represented by dents and fillings in the middle or terminal portion. The con-

Fig. 44.1 Pulmonary P wave and mitral P waves.



The concept of QRS duration has lost value for the diagnosis of block compared to the morphological concept, since there are true blocks with typical morphology in which the QRS duration is delayed only in a minimal fraction of time (in incomplete blocks).

3. Repolarization alterations, which consist of a negative slope of the ST segment, with superior convexity and negative and asymmetric T waves, in the leads that face the ventricle that has the blocked branch.

The concept that conduction disorders through the bundle branches were associated with the deviation of the QRS electrical axis to the right in cases of complete right bundle branch block (CRBB), or to the left in cases of complete left bundle branch block (LBBB), changed after the observations of Dr. Mauricio Rosenbaum from the Argentine school of cardiology:

right tracings in the first precordial deviations and left, in the standard leads; In some patients the electrical QRS axis is sometimes deviated to the right and other times to the left. From this arose the concept called by Rosenbaum: anterior, posterior and intermittent left hemiblocks.

The clinical significance of bundle branch blocks in general is variable; These can be seen in people with apparent good health, as occurs in right bundle branch blocks, and other times they manifest the existence of an evolving injury, or ischemic heart disease, as occurs in most cases of left bundle branch block.

Classification

Branch blocks can be: transient, as an expression of an acute condition (pulmonary thromboembolism and cardiac infarction); intermittent, typical of extrasystoles (premature supraventricular or atrial contractions) and tachyarrhythmias (atrial fibrillation and flutter); and permanent, due to pathophysiological interruption of one of the branches of the bundle of His (cardioangiosclerosis, infarction and trauma).

Of branch locks, we will only study:

Complete right bundle branch block (BCRD).

- Complete left bundle branch block (LBBB).
- Hemiblocks or fascicular blocks.

Complete blockage of right branch to BCRD

Electrocardiographic criteria (fig 44 2)

1. QRS complexes. Duration equal to or greater than 0.12 s.
2. rSR' complex in V,, V, and V, (looking like M).

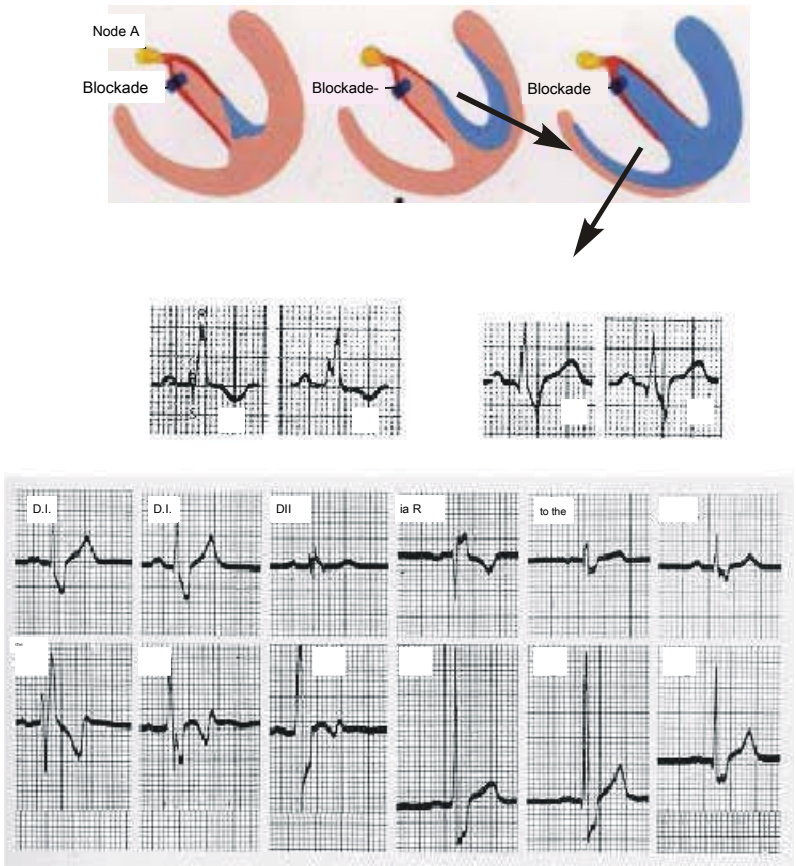


Fig. 44.2 Complete right bundle branch block.

3. Morphology. Late and stuck S waves in DI, DII, aVL, V, and V, as a reciprocal or mirror image. Late R in aVR.

4. Electrical QRS axis. It can be within normal limits, that is, between -30° and +90°, although sometimes it can also be deviated to the left or right if the blockage is associated with other alterations.

Semidiagnosis

1. BCRD can be observed in both right and left ventricular diseases.
2. Sometimes it is mainly associated with coronary heart disease, cardiomyopathies and myocarditis.
3. In atrial septal defect (AC) it may be an expression of diastolic overload of the right ventricle.

4. It can be found in individuals without heart disease.

Incomplete or minor right bundle branch blocks are very common. Its electrocardiographic criteria are given by the morphology of the right bundle branch block, but the QRS duration should not reach 0.12 s.

Complete blockage of left branch BCRI

Electrocardiographic criteria (fig 44 3)

A. In precordial leads.

Wide QRS complexes: 0.12 s or more in duration, made up of R waves indented in V, and V; Opponent ST (negatively shifted), negative T waves.

Two types of alterations can also be observed in the right precordials, as a reciprocal mirror image: notched QS from V, to V,, with elevated ST and positive T wave, or rS complex in the first precordials, from V, to V,. Both types with elevated ST and positive, asymmetric T waves.

B. In standard leads, wide QRS, 0.12 s or more in duration.

In DI:

1. R notched without q or s.
2. ST displaced downward and straight, forming a body with the T wave, which is negative asymmetric.

In DIII the mirror image of DI is observed:

1. So QS nicked.
2. There may be small initial r wave (rS complex).
3. ST shifted up.
4. T positive, asymmetric.

In DII, which is nothing more than the sum of DI and DIII, the QRS morphology will be related to the degree of left axial QRS deviation.

C. In unipolar limb leads.

1. In aVL, R wide and notched, a small q may appear. ST segment displaced downward with asymmetric negative T wave (pattern almost the same as in DI).

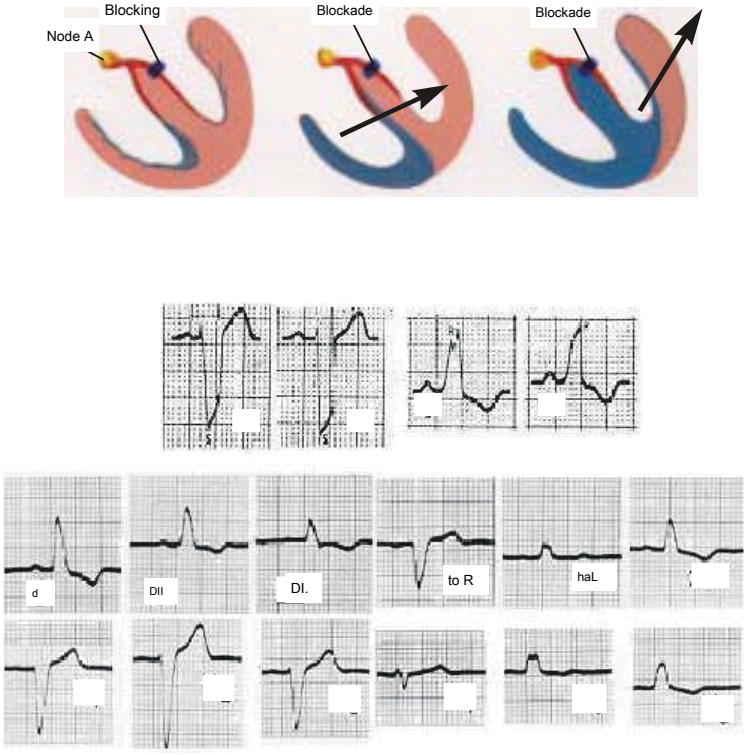


Fig. 44.3 Complete left bundle branch block.

2. In aVF the mirror image of aVL is observed. So deep and indented QS with elevated ST and asymmetric positive T wave.
3. In aVR the QRS is almost always negative.

Let us remember that there is never a q or s wave in DI, nor in V, and V, and if they are observed they may be due to the coincidence of old anteroseptal infarcts.

Semidiagnosis

1. Ischemic heart disease.
2. Hypertensive heart disease, with previous left ventricular growth.
3. Primary and secondary infectious cardiomyopathies.
4. In the course of luetic aortic insufficiency.

Complete blocks of the left bundle branch, from an electrocardiographic point of view, can be divided into: truncal and divisional, according to the anatomical and functional conception of the bundle of His and whether the injury is to the branch or trunk or to some of its divisions (posterior or anterior fibers).

Truncular blocks are related to ischemic heart disease, and divisional blocks to muscle diseases, such as cardiosclerosis and cardiomyopathies.

Incomplete or minor left bundle branch blocks are highly discussed. The alterations found, QRS duration not very long (does not exceed 0.09-0.1l s); It could correspond to a certain degree of left ventricular growth.

emiblo ueos or fascicular blo ues

As we have said, the term hemiblock was created by Rosenbaum; in accordance with the new anatomohistological and functional conception of the bundle of His already described in Section I, in Chapter 10 of this However, the most appropriate name is fascicular block.

Concept

Because the right branch and the anterior division of the left branch of the bundle of His are the most vulnerable, their injuries or alterations are the ones that most frequently cause fascicular blocks.

Classification

The fascicular blocks that can occur are:

a) Blockages of a fascicle:

- Anterior left fascicular block.
- Posterior left fascicular block.

b) Bifascicular blocks:

Right bundle branch block with anterior left fascicular block.

Right bundle branch block with posterior left fascicular block.

Incomplete left bundle branch block with anterior left fascicular block.

- Incomplete left bundle branch block with posterior left fascicular block.

c) Trifascicular blocks.

We will describe only a few, of greater importance clinic:

Anterior left fascicular block

Also called anterior divisional block or left anterior hemiblock (Fig. 44.4).

Electrocardiographic criteria

1. QRS electrical axis between -30° and -90° 2. Morphology q-I, S-III.
3. Duration of the QRS complex up to 0.10 s.

Before the knowledge of fascicular blocks, these tracings were considered normal with left axial deviation.

Semidiagnosis

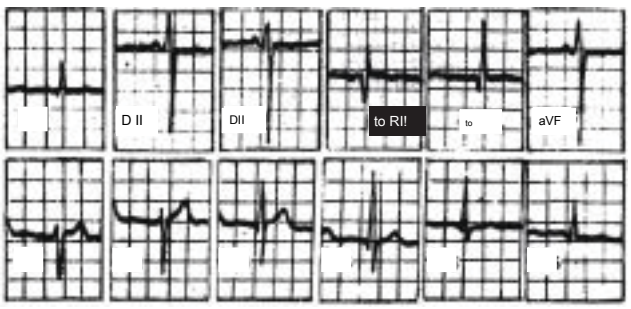
1. Anginous syndrome, confirming coronary insufficiency, in addition to indicating which of the coronary arteries is damaged: the anterior descending, branch of the left coronary artery.
2. Patients with diastolic or left ventricular volume overload, in those affected by aortic diseases.

Right bundle branch block with left anterior fascicular block

Electrocardiographic criteria (fig. 44.5)

1. QRS electrical axis to the left between -30° and -180.
2. Morphology q-I, S-III.

Fig. 44.4 Left anterior fascicular block.

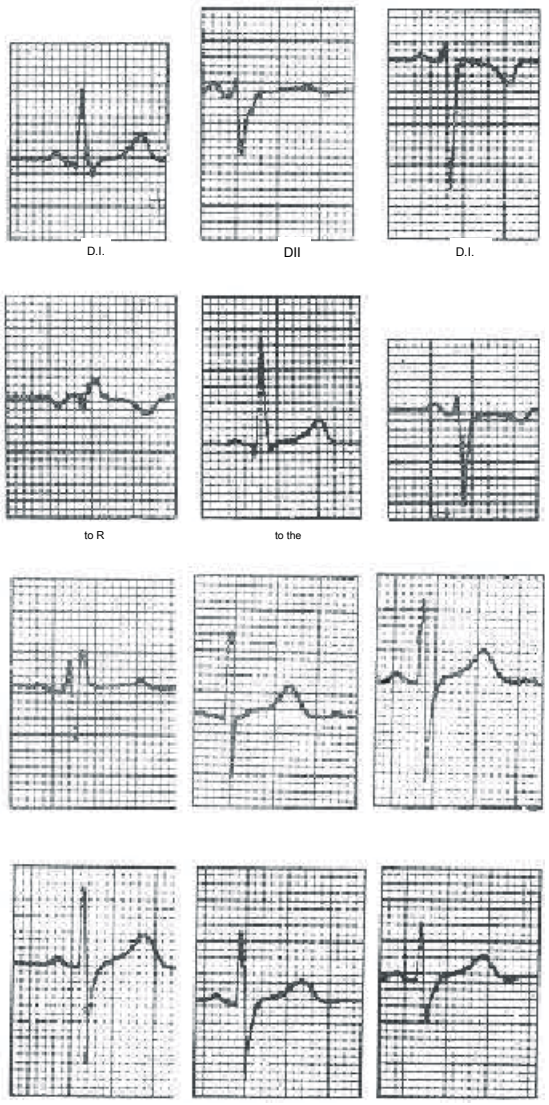


3. Late S in DI, aVL, V, and V
4. rsR complex in V,;
5. QRS duration between 0.10 and 0.12 s, in incomplete right bundle branch block and equal to or greater than 0.12 s in complete block of the same branch.

Semidiagnosis

1. Congenital heart diseases: atrioventricular comunis and ostium primum.
2. Acquired heart diseases: diphtheria myocarditis, aortic valve disease, septal or anterolateral infarction, primary cardiomyopathies and sclerodegenerative processes of the conduction system in the elderly.

Fig. 44.5 Right bundle branch block with left anterior fascicular block.



Trifascicular block

Its diagnosis is suspected in the classic electrocardiogram, indirectly, due to the presence of bifascicular block with a prolonged PR interval, as an expression of first-degree atrioventricular block. Direct diagnosis is made through an electrocardiogram of the Hiz bundle (hisiogram).

We should not conclude this section on fascicular blocks without first pointing out that, for some authors - among them Narula - the Rosenbaum criterion is not solid. They base this on the fact that electrophysiological studies demonstrate conduction defects in the three fascicles and it would be more appropriate to call them partial bilateral blocks.

ALTERATIONS OF THE RS IN HEIGHT

QRS alterations in height can be due to excess or defect. An ECG with larger than usual QRS complexes is a criterion for ventricular growth and with very small QRS complexes, a criterion for microvoltage.

GROWTHS 0 VENTRICULAR IPERTROPHIES

Ventricular growth first manifests itself as dilation and after a certain time the muscle hypertrophies. That is why the term "ventricular growth" is preferred to that of " ventricular hypertrophy", because the former includes both dilation and hypertrophy, although here we will sometimes use them interchangeably, to refer to both pathophysiological phenomena.

Electrocardiographic criteria

1. QRS morphological criteria.
2. QRS voltage indices.
3. Duration of the QRS complexes and intrinsicoid deflection.
4. Associated criteria for ventricular overload.

The morphological criterion is very important and is related to the leads that capture the electrical phenomena of the free wall of the ventricle and to the electrical QRS axis; The alterations recorded in these leads, including the morphological criteria of overload, especially systolic, allow us to suspect the diagnosis (see below).

In the study of hypertrophies, voltage is very useful. The most commonly used voltage indices are the Sokolow index (right and left), the Cornell index for left growths, and the White-Bock index. The Sokolow index is sought in the precordial leads.

Left Sokolow. Add the S major of V, or V, and the R major of V, or V

Normal: less than 35 min. Equal to or greater than 35 mm indicates left ventricular growth.

Right Sokolow. Add the R in V, and the S greater than V, or V

Normal up to 11.5 mm. Greater than 11.5 mm indicates right ventricular growth.

Cornell index. Add the R in aVL and the S in V,.

Normal up to 20 mm in women and 28 mm in men.
Greater than these figures indicates left ventricular growth.

White-Bock index. It is studied in the standard leads DI and DIII using the formula: (R in DI + S in DIII) – (R in DIII + S in DI).

Normal index between -15 and +30. Indices less than -15 indicate right ventricular growth and greater than +30, left ventricular growth. A normal index does not exclude the possibility of ventricular growth.

The duration of the QRS complex, in ventricular growths, should be greater than 0.08 s (without reaching 0.12 s) in the leads facing the left or right ventricle, depending on which hypertrophy occurs.

The intrinsicoid deflection time of QR or R in the lead in front of the hypertrophied ventricle should be increased by more than 0.035 s in V,, and by more than 0.045 in V.

The electrical[6] QRS axis will generally be deviated toward the hypertrophied side.

To be more explicit about the morphological criterion, let us point out that in left ventricular growth (fig. 44.6) we find, in standard and unipolar limb leads, patterns with a strong positive predominance with alterations in repolarization: R in DI and aVL; while leads DIII and aVF show strongly negative patterns: rS.

Variations that occur depending on the anatomical position of the heart can make diagnosis difficult.

In left precordial leads (V, V, V) there is large R amplitude, slightly negative ST segment and negative T wave (and q wave in 50 9% of cases); there are transitional complexes in V, and V,; in right precordials, V, and V,, the complexes are rS and sometimes QS.

In right ventricular growth (fig. 44.7) we find, in the limb leads, a strong positive predominance in those that capture the potentials of the lower portion of the body (DII, DIII and aVF) and complexes with a strong negative predominance in DI and aVL; qR in DII, DII and aVE, rS in DI and aVL, T wave with a tendency to invert in relation to QRS and R wave in aVR.

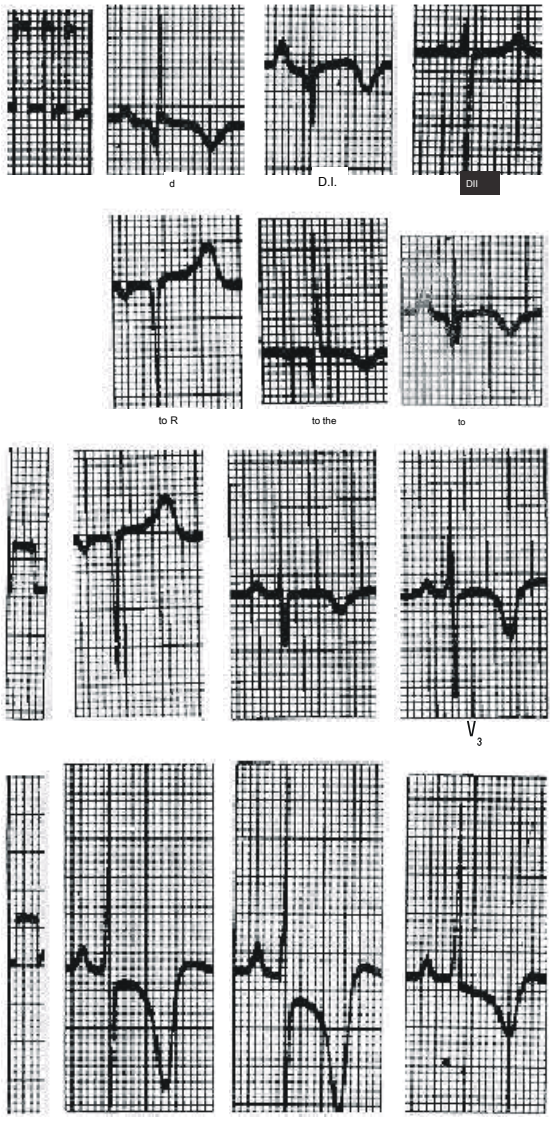


Fig. 44.6 Left ventricular growth.

In right precordial leads (V, V, and Vr) there is a predominance of positive waves type RS, Rs, rsR (R equal to or greater than S), sometimes preceded by a q wave; negative ST segment elevation with negative T waves, the more marked the hypertrophy, the further to the left it extends, and can reach up to V. A negative T wave in V, can be normal at any age, but if it also appears in V, and V, in an adult man, then it can be pathological in a high percentage of cases.

It must be remembered that the normal Tnegative is asymmetric.

In left precordial leads (V, and V) deep S waves or normal type patterns may appear, sometimes QS in V.

Right ventricular hypertrophy is frequently associated with pulmonary-type P waves or complete or incomplete right bundle branch block.

The ventricular growth criteria are necessarily linked to those of overload, since dilation

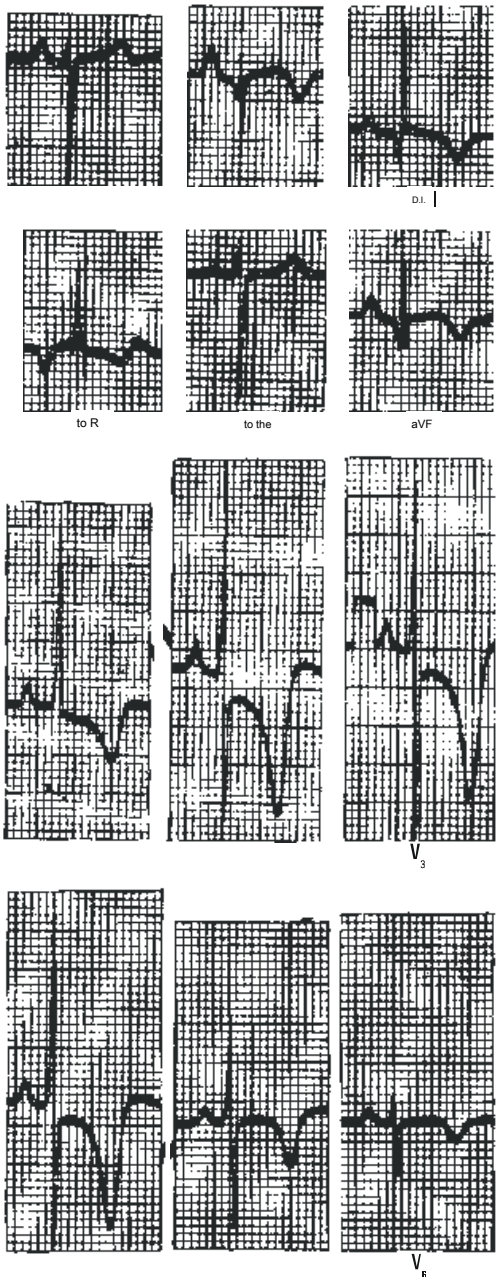


Fig. 44.7 Right ventricular growth.

occurs as a need for diastolic overload and hypertrophy, as a need for systolic overload. For this reason, we will first focus on the concepts of overloads and then explain each one of them, although systolic overloads appear on the ECG as ST-Ty alterations, not QRS.

Concepts of ventricular overloads

It is demonstrated by the work of several researchers, among them, mainly, those of Professor Enrique Cabrera, that the heart adapts to the load imposed on it, according to physical laws and principles. Thus, we can see that in certain valvular diseases events occur that They allow us to understand this adaptation. Aortic stenosis causes work overload to the left ventricle, and aortic insufficiency causes volume overload to this ventricle. Work overload was called systolic overload or late overload by Cabrera (it is also known by the terms pressure overload and concentric overload), and volume overload was called diastolic or initial overload.

For a better understanding of these concepts, Figure 44.8 represents a phonocardiogram and an electrocardiogram where the electrical and mechanical phenomena of cardiac activity have been indicated.

Left ventricular systolic overload

If we take aortic stenosis as an example, we see that due to the systolic overload that it causes, the left ventricle adapts by increasing the work (contractility), and with greater work, greater thickness of its walls, hypertrophy is produced and there is greater oxygen consumption, this mechanical phenomenon having repercussions on the electrocardiogram, mainly in the phase of ventricular repolarization, on the ST segment and the T wave.

The electrocardiographic manifestations will be given by inversion of the T wave, and the ST segment moves downward, straight or convex, forming a body with the T wave, giving it a characteristic mnorphology: inverted, with its first straight or flat branch and the second curved branch, unlike coronary ischemic T, also inverted, but in which its two branches are symmetrical. There is also high left ventricular systolic overload. These alterations are seen in the left precordial leads (V, V) (see Fig. 44.6).

In left ventricular systolic overload, the QRS complex of the electrocardiogram may be normal in height, because the hypertrophy is concentric, involving the septum and large septal depolarizations, which have the opposite direction and direction.

Semidiagnosis

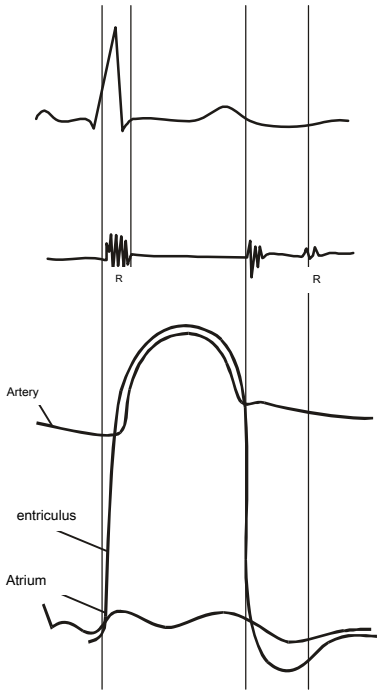
1. High blood pressure.
2. Aortic valve stenosis.
3. Aortic coarctation.

Right ventricular systolic overload

It is due to pathological situations in which the right ventricle has to overcome great resistance during systole, which causes it to develop enormous pressure and hypertrophy its walls.

The electrocardiographic signs of right systolic overload are those of ST-T, associated with those of right ventricular growth which, as we saw previously, is

Fig. 44.8 Simultaneous phonocardiogram and electrocardiogram.



They are given by QRS patterns with a strong positive predominance, which correspond to flattened T waves or negative convex shift of the ST with asymmetric inverted T waves, in leads DII, DII, aVE, V, and V, which are those that face said ventricle. The electrical axis of QRS is always on the right and its duration can reach up to 0.10 s. As systolic overload increases, the R waves become more positive and the T waves more inverted, they can extend up to V, and sometimes become symmetrical (the so-called Zuckerman ischemic type T). The QRS complexes will be: rsR, with the R in V, higher, cleaner and its voltage 15 mm or greater, rS complexes in DI and aVL, and qR complexes in DII, DIII and aVF (see Fig. 44.7).

Semidiagnosis

1. Congenital heart diseases: isolated pulmonary stenosis, ventricular septal defect (VSD) with pulmonary hypertension and patent ductus arteriosus with pulmonary hypertension, Eisenmenger syndrome and complex, and tetralogy of Fallot.
2. Acquired heart diseases: essential or primary pulmonary hypertension, chronic cor pulmonale and mitral stenosis with pulmonary hypertension.

Left ventricular diastolic overload

In diastolic overload (initial volume or eccentric overload) of the left ventricle, from its Initially, the ventricle handles a greater volume of blood than normal.

Taking aortic valve insufficiency as an example, we can understand that the left ventricle during diastole, in addition to receiving the blood that normally arrives from the left atrium, also receives that which arrives abnormally due to the insufficiency of the aortic sigmoid, for which it handles an abnormal volume of blood, and when it dilates it compresses, due to its diastolic enlargement, the left branch of the bundle of His. When ventricular activation (QRS) begins, it occurs with a delay in conduction, which causes a deep Q wave called hemodynamics in the electrocardiogram, without changes in the T wave.

These alterations are seen in leads V, and V,, which reflect the free wall of the left ventricle.

Semidiagnosis

1. Aortic insufficiency.
2. Mitral insufficiency.
3. Patent ductus arteriosus, in its first phase, without pulmonary hypertension.

Mixed or combined overload of the left ventricle

Systolic overload of the left ventricle occurs after the ventricle hypertrophies (compensation mechanism), which causes relative anoxia of the cardiac muscle (which brings with it major problems in the contractility of muscle fibers) which resorts to diastolic overload to defend itself.

Semidiagnosis

In addition to being present in the aforementioned cases of large left hypertrophies of any etiology, mixed overload can also be seen in patients with a double rheumatic aortic lesion.

Right ventricular diastolic overload

The right ventricle responds to diastolic overload by dilating its walls, which causes the right bundle branch block (rSR) pattern to appear in the leads that face the right ventricle (V,, V,). This occurs due to elongation or compression of the right branch of the bundle of His, located on the endocardial surface of the right ventricle, as it dilates. The pattern that appears on the right ventricle is, we repeat, rSR, which is not pathognomonic of diastolic overload of the right ventricle, nor does it always mean dilation.

MICROVOLTA E

An electrocardiogram is considered to have a low voltage criterion when the largest QRS deflection at the

least one of the six limb leads is less than 5 mm (0.5 mV) and is less than 10 mm in all six precordial leads.

The voltage decreases when poor conductive substances are interposed between the heart and the electrodes, such as air in emphysema; fluid in pericarditis with effusion and fat in obesity. Microvoltage is also observed in myocardial infiltrations such as myxedema, avitaminosis such as beri-beri, and in large myocardial necrosis.

PRESENCE OF PATHOLOGICAL

MI0C RDICA NECROSIS

The criteria for pathological Q wave due to myocardial necrosis are:

1. Q wave lasting more than 0.03 s.
2. Q wave with a depth greater than 30 9% of the Ro height of 25% of the entire QRS.
3. Presence of OS waves. If they are in the right precordial leads (V,, V, and sometimes V.), there should be absence of complete left bundle branch block.
   Some cases of left ventricular hypertrophy, or left bundle branch blocks, may be confused with an anteroseptal infarction. In these cases mentioned, they are accompanied by a positive T wave.
4. The dent of the QS wave that appears in the right precordials, especially if it is a dent in its ascending branch, and produced more than 0.05 s after the complex begins, is also considered a sign of myocardial necrosis (Cabrera and Friendland sign).

We believe it is convenient to point out here that whenever a wave appears, no matter how small, in right precordial leads, its disappearance in another lead taken further to the left is always pathological and suggests infarction.

In very horizontal hearts, the Q wave in DII can be very marked. In these cases, it is modified when that derivation is recorded at the moment the subject is ordered to take a deep inspiration (DIII inspired). This abnormal Q wave of DIII will not appear in aVF or DII if it is only a horizontal heart.

The topographic or localization diagnosis allows us to specify the face or wall on which the infarction is located (in this case the recording of the Q wave of necrosis), which we can fundamentally locate in four projections:

- From the front.
- Posterior or diaphragmatic face.
- Septal (anteroseptal or posteroseptal).
- On the left side.

To make the topographic diagnosis of the infarction, it is necessary to know the regions of the epicardial surface that each electrode explores.

Lead DIL, as well as leads DII and aVF, face the diaphragmatic or posteroinferior wall of the heart. Thus, a pathological Q in these leads means diaphragmatic or posteroinferior necrosis.

The leads that face the lateral wall of the left ventricle are aVL, DI and V. Lead DI faces the lower part of the lateral wall, in a frontal plane and V, in the transverse plane, while aVL faces the highest lateral part of said ventricle. Thus, a pathological Q wave in these leads means high lateral necrosis, in the last one, and lateral necrosis in the first two.

V, faces the septum on its anterior part. A pathological Q in V means septal necrosis. If the pathological Q appears in leads V, V, and V, which face the septum, as well as the anterior surface of the heart, the necrosis will be anteroseptal.

Leads V, V, and V face the anterolateral wall of the left ventricle, so the presence of a Q for necrosis in them will mean anterolateral necrosis.

When the myocardial infarction is not old, it presents other electrocardiographic alterations, in addition to the Q for necrosis, in the same leads where it is located, which allows the evaluation of the evolutionary stage of the infarction and its topographic diagnosis, as will be seen later.

DIASTOLIC OVERLOAD WAVE OF THE FIRST VENTRICLE

As expressed in the analysis of overloads, the observation of a deep Q wave in V, and V, in the presence of left ventricular growth, without negative alterations of the ST and T, is an element suggestive of diastolic overload of the left ventricle and not of myocardial necrosis.

ST AND T WAVE ALTERATIONS

SECONDARY ALTERATIONS OF VENTRICULAR REPOLARI ATION

Secondary ST-T alterations consist of a convex negative shift of the ST, with an asymmetric negative T wave, in the leads facing the altered ventricle, and mirror images or re-

cyprocal, positive displacement or concave ST elevation, with asymmetric positive T, in the leads facing the opposite ventricle.

These ST and T wave alterations may be secondary to:

- Complete branch blocks.

-Ventricular systolic overloads.

When we studied complete bundle branch blocks, we already stated that these are accompanied by repolarization disorders secondary to the block, consisting of a convex ST depression with asymmetric negative T, in the leads that face the blocked branch, and the opposite, like a mirror image, in the opposite leads. So, if a complete bundle branch block is detected when studying the QRS, these alterations in ventricular repolarization are secondary to the block and nothing else.

The same ST-T electrocardiographic pattern can be observed in ventricular growths, when there is systolic overload of the enlarged ventricle, with its mirror image in the leads facing the opposite ventricle. These alterations are not reported as secondary repolarization alterations, as in blockade, but rather have their own meaning: the existence of systolic overload of the enlarged ventricle.

PRIMARY ALTERATIONS OF VENTRICULAR REPOLARI ATION

Primary alterations of ventricular repolarization (ST-T) are mostly due to myocardial ischemia. As these alterations vary and are accompanied or not by others, depending on the type of ischemia produced, we consider it appropriate to explain the classic types of painful ischemic heart diseases, with the electrocardiographic disorders associated with each of them.

Is umic heart disease

Concept

They are due to a deficit in the amount of blood due to a sudden interruption of coronary flow, or also to an oxygen deficit, which can be temporary or persistent. When any of these conditions persist, modifications occur in the cells and structures of the heart that go through several successive stages according to the time that has passed.

When ischemia or anoxia is transient, the clinical picture is that of angor pectoris or angina pectoris; When it is prolonged, myocardial infarction (heart attack) appears.

We will review acute, transient and permanent coronary insufficiency, and chronic coronary insufficiency.

Classification

A. Transient acute coronary insufficiency.

- Typical angina: Heberden.
- Atypical angina or variant angina: Prinzmetal.
- Subendocardial ischemia.

B. Permanent acute coronary insufficiency.

- Myocardial infarction.

C. Chronic coronary insufficiency.

Acute transient coronary insufficiency

Electrocardiographic criteria

Ischemic signs are:

1. Flattened or inverted T waves in those leads that face or capture the potentials of the epicardial surface of the ischemic area.
2. If a component of injury is added to the ischemia, the complexes, apart from presenting T inversion, will show a slight positive ST segment elevation.
3. Mirror image, in those leads that face or capture the potentials of the area opposite the ischemic area.

The ischemic manifestations of angina pectoris, depending on their location, can be classified as:

- Ischemia of the anterior face.
- Posterior face ischemia.
- Subendocardial ischemia.

All offer electrocardiographic signs in the leads facing the ischemic zone and the mirror image in the leads opposite said zone.

Typical angina: Heberden. ST segment displaced negatively (downwards) in the precordials V, V, and V, with positive T. In aVR, elevated ST with negative T.

Signs of ischemia can also be given by flattened or inverted Ts and a slight positive ST slope in the leads facing the area of the affected ventricle, depending on their location in the thickness of the ventricular wall with mirror images in the leads opposite the ischemic area.

These signs of ischemia do not have to appear in all leads, as they depend on its greater or lesser extension.

Atypical angina or variant angina: Prinzmetal. The pain is experienced by the patient at rest, and the electrocardiographic criteria are characterized by: monophasic waves in DII, DIII and aVF; The route returns to normal in hours or days. As can be seen, the location of these alterations correspond to areas of the

inferior or diaphragmatic surface of the heart supplied by the right coronary artery.

We point out that the appearance of electrocardiographic signs that only appear during the crisis and return to the previous characteristics of the tracing after rest is a fact of extreme importance in the electrocardiographic diagnosis of angina pectoris, whatever its type.

Subendocardial ischemia. It is characterized by signs of ischemia in aVR, which is the shunt facing the endocardial surface of the heart. The mirror image varies depending on the location of the ischemia, but above all, in leads V, V, and V.

We must remember that the shunt to VR normally presents an inverted T wave, so to suspect the existence of a subendocardial ischemia, this inverted T must be accompanied by a positive ST segment elevation, as well as have a coronary morphology (symmetrical). The presence of a mirror image has a lot of value in this case.

Stress tests (Master test and ergometric test)

When studying angina pectoris, we pointed out the possibility that patients with an anginal syndrome presented a normal electrocardiogram, which is interpreted as that these patients have effective coronary circulation at rest, but nevertheless, they have a highly compromised coronary reserve and this deficit manifests itself when ischemia appears when myocardial demands increase during effort. That is why these patients are subjected to a stress test, which can consist of a classic Master's test or an ergometric test, either with a bicycle or with a rolling mat.

The Master test consists of performing an electrocardiogram on the patient at rest; Then you are asked to make a certain effort and another electrocardiogram is immediately performed; In this way, alterations not obtained in the previous layout are captured. In the ergometric test, the ECG is recorded at rest, during and after exercise, and pulse rate and blood pressure are simultaneously monitored.

It is important to note that the effort made must be evaluated with a series of data: patient age, weight, etc.

However, in a general way it can be said that the classic Master test consists of making the patient climb two steps, each approximately 20 cm high, at a pace that allows him to do so for about 25 cm.

times in a time interval of approximately 90 s.

The test should always be performed with great caution, since it can cause a heart attack and even sudden death.

Positive electrical signs of a stress test

1. ST depression of 1 mm or more, 0.08 s after the J point, using the PQ segment as a baseline in six consecutive cycles.
2. Appearance of the already known signs of ischemia, in any of its locations, especially the subendocardial.
3. Negative T waves that become positive, and this should not be mistakenly interpreted as a favorable action of the effort and that a predominant subendocardial ischemia has appeared.

Permanent acute coronary insufficiency. myocardial infarction

Heart attacks almost always occur in the left ventricle. If they occur on the lateral aspect of the right ventricle, which is rare, they are impossible to diagnose by electrocardiogram.

The electrocardiogram of myocardial infarction allows three types of diagnoses to be made:

- Positive diagnosis.
- Topographic or location diagnosis.
- Evolutionary diagnosis.

The positive diagnosis is made when obvious signs of infarction are observed in the electrocardiogram.

These are:

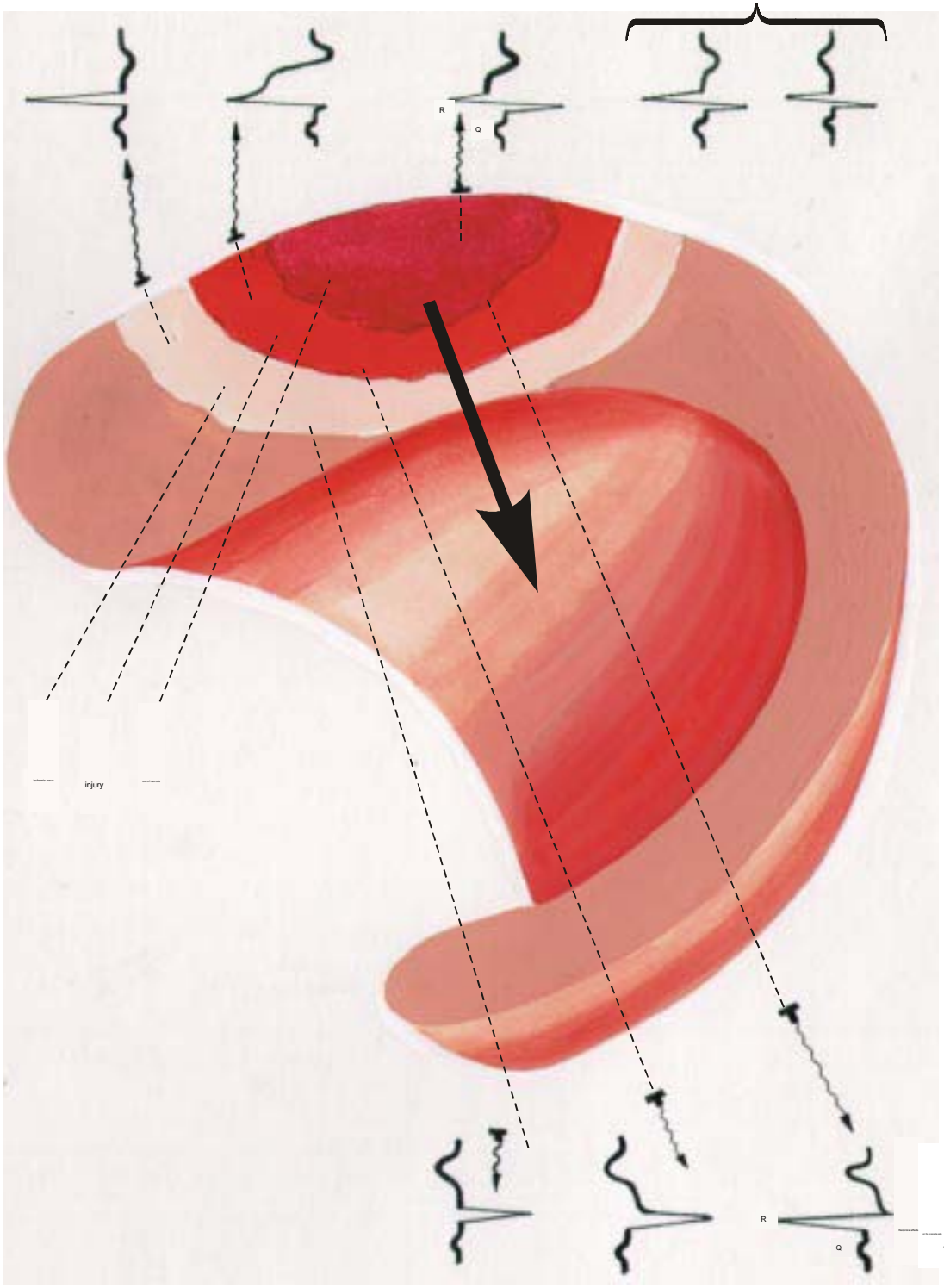
1. Pathological Q wave (necrosis).
2. Positive shift of the ST segment (lesion).
3. Inverted T wave (ischemia).

These signs of infarction appear in the leads that collect or capture the potentials of the infarcted area and give a typical morphology to the tracing (fig 44.9).

In the leads that face the area opposite the lesion, the so-called mirror image appears (fig. 44.10), which consists of:

1. High R waves (sometimes).
2. Negative shift of the ST segment.
3. High positive T waves.

Although necrosis is a subsequent stage that follows ischemia, the pathological Q wave usually presents within the first hours in the case of infarction. In those cases where it is not yet present, the


R
Q
injury
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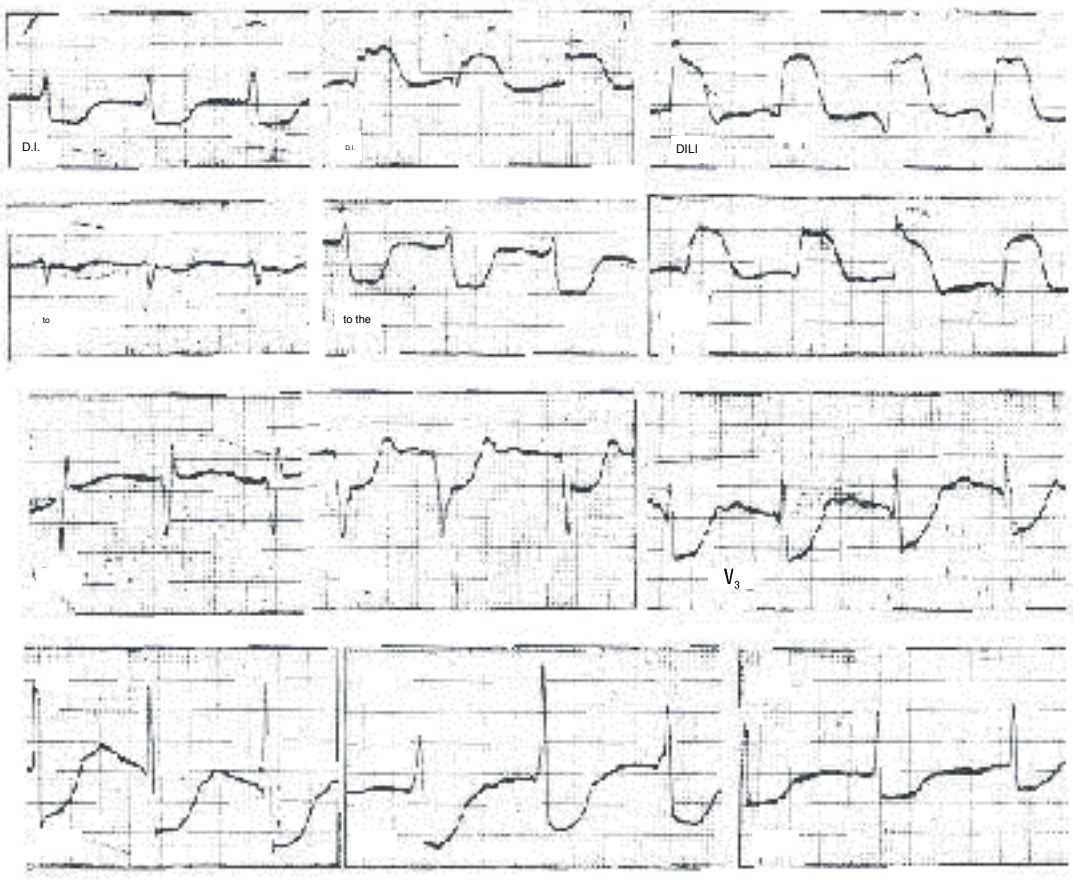


Fig. 44.10 Mirror image of a posterior infarction.

Marked alterations of the ST segment and the T wave, together with the clinical manifestations, will allow the diagnosis to be suspected.

Regarding their extension in the thickness of the face or wall, infarcts can be:

1. Subendocardial.
2. Intramurals.
3. Transmurals.
4. Subepicardial.

Topographic classification of heart attacks

As explained when studying the pathological Q, according to the leads where the electrocardiographic signs of infarction are found, a topographic diagnosis can be made.

	ì Anteroseptal
art a posteriore	í Anterolateral
	1 Previous extensive
lateral art	
	ì Posteroinferior
arto p0 posteriore	1 Posterolateral
	1 Posterosuperior
arto eptal pro u do	

Previous heart attacks. They are caused by obstruction of the left coronary artery or one of its branches, the anterior descending or the circumflex. They affect the left ventricle on its anterior and lateral surfaces, and to a very small extent the anterior surface of the right ventricle; Since the anterosuperior portion of the septum is irrigated by the left coronary artery, it can also become infarcted due to occlusion of this coronary artery.

Anteroseptal: it is located on the anterior surface of the heart in the area located in the vicinity of the septum and extends approximately over the anterior surface of the right and left ventricles, as well as over the anterosuperior portion of the septum. The electrocardiographic signs of infarction, described in the positive diagnosis of infarction, will appear in leads V.,, V, and V,. Lead V may also be altered. Leads V and V appear normal. The limb leads will not present alterations, perhaps only a flattened T in ID (Fig. 44.11).

Anterolateral: the anterior wall and the lateral wall of the left ventricle are affected; The anterior portion of the septum remains intact. Signs of infarction appear, preferably, in the left precordial leads V, and V, and generally extend to the area

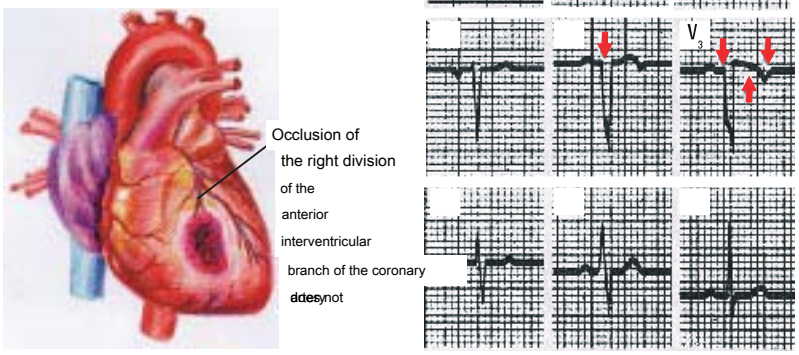


Fig. 44.11 Anteroseptal infarction (V-shaped, and V).

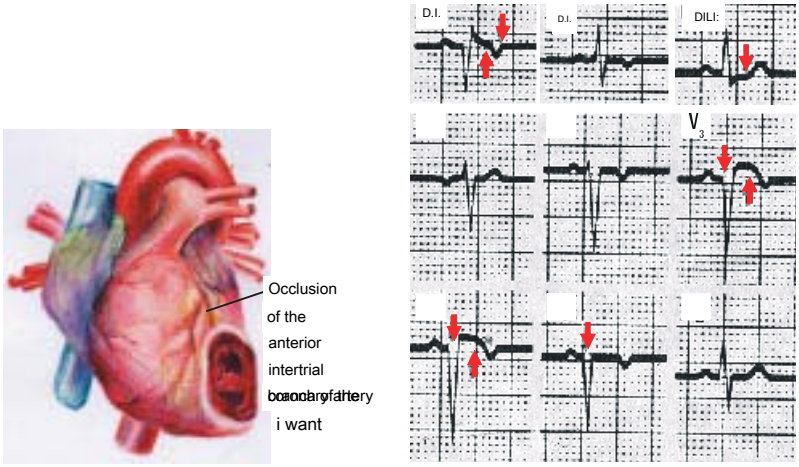


Fig. 44.12 Anterolateral infarction (in DI, DIII, V, V, and V).

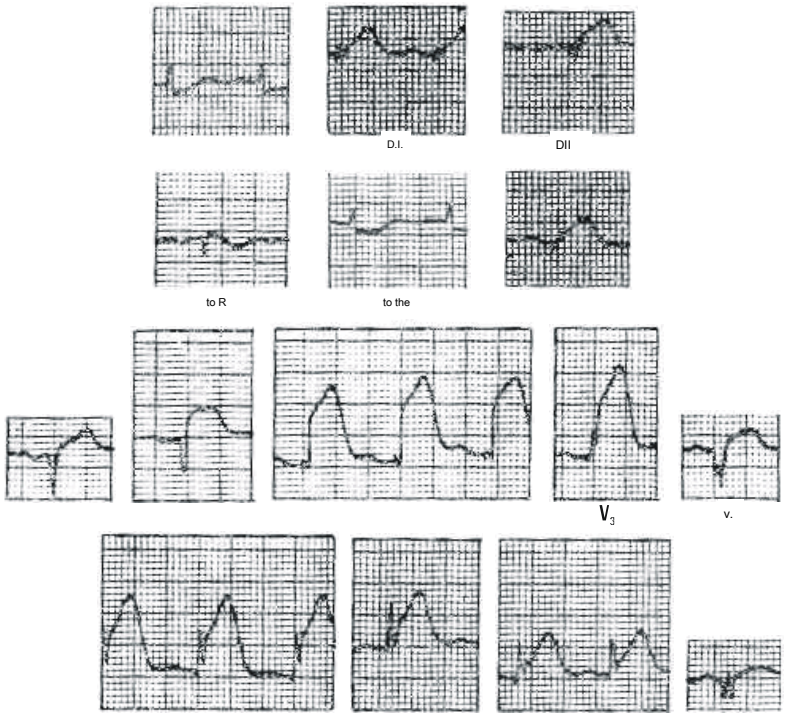


Fig. 44.13 Extensive anterior infarction (see appearance of and decrease in ST elevation in V,, V, and V,, 18h later).

of transition V, and V, and also appear in the limb leads DI and aVL (fig. 44.12).

Extensive anterior: it can be considered as the sum of the two previous ones (fig. 44.13). Signs of infarction will appear in all precordial leads, from V to V. The extension towards the lateral wall of the left ventricle causes these signs to also manifest in DI and aVL.

Lateral infarction. This infarction affects the lateral wall of the left ventricle in its upper, anterobasal portion (fig. 44.14). Signs of infarction appear in leads DI and aVL, but are generally not shown in precordial leads. They may, however, appear when performing high precordial derivations, at the level of the second and third left intercostal spaces, in the midclavicular or anterior axillary line.

Subsequent infarctions. They are due to occlusion of the right coronary artery, which largely supplies the posterior surface of the heart, the rest of which is supplied by the circumflex branch of the left coronary artery. In these infarctions, rhythm disorders occur very frequently, such as: intense bradycardia, different degrees of atrioventricular block, etc., since the right coronary artery is the one on which the irrigation of a large part of the heart's conduction system, mainly the sinus and atrioventricular nodes, fundamentally depends. This fact makes these heart attacks have a reserved prognosis.

Posteroinferior: the infarct covers a portion of the posterior wall of the left ventricle (Fig. 44.15). Signs of infarction will appear in leads DII, DIII and aVF, and the mirror image is collected in some of the precordial leads.

Posterolateral: due to occlusion of the circumflex artery, and extends to the lateral wall of the left ventricle (Fig. 44.16). Signs of infarction appear in leads DII, DIII, aVF, V, and V

Posterosuperior: it is generally small, and is not detected in the classic leads of the electrocardiogram; It appears only in esophageal diversions, which are not routinely performed (Fig. 44.17). However, the reciprocal mirror image can be seen, on the anterior surface, in the precordial leads V, and V, and sometimes in V,, consisting of a small and wide R, negative concave displacement of the ST and positive T, which correspond to the mirror image of the q of necrosis, the pattern of injury of the ST and the negative T of ischemia, respectively.

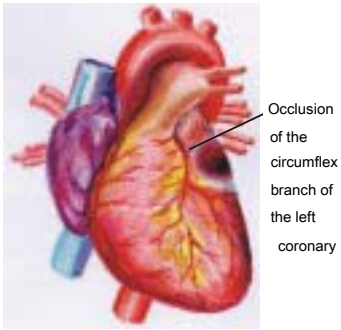
Vainly.

Deep septal infarction. It massively affects the septum and extends to the anterior and posterior aspects of the heart (Fig. 44.18). Signs of infarction therefore appear in leads DII, DIII and aVF, as well as in leads V, V, and V, and can extend to the left precordial leads V, V, and V.

Remembering the classification relative to its extension in the thickness of the face or wall, we will point out:

Subendocardial infarction. It is a rare type of infarction, which is limited to the subendocardial layers of the left ventricle. Signs of infarction appear in lead aVR, which explores the potentials of the cavity. Since the aVR lead under normal conditions can present a large Q wave, as well as an inverted T, only a strong positive slope of the ST segment will be significant, without which the pathological Q wave criterion cannot be established. The precordial leads will show the mirror image and negative shift of the ST segment with strongly positive T waves; The image of infarction on the posterior surface does not appear in DII, DIII and aVE.

In short, the subjective symptoms, clinical signs and humoral picture give the diagnosis, since the electrocardiogram shows only a subendocardial ischemia.



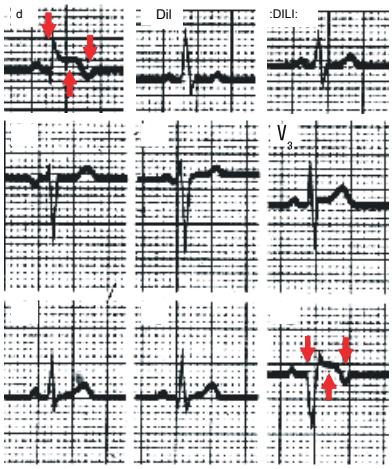


Fig. 44.14 Lateral, anterobasal infarction (in DI and V).

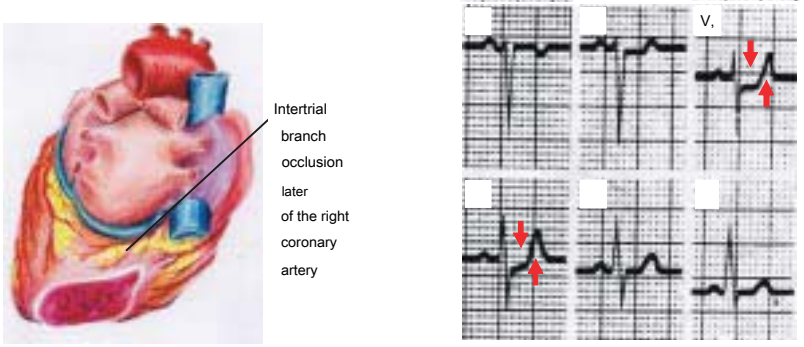


Fig. 44.15 Posteroinferior infarction (in DII, DIII and aVF; mirror image in DI, V, and V).

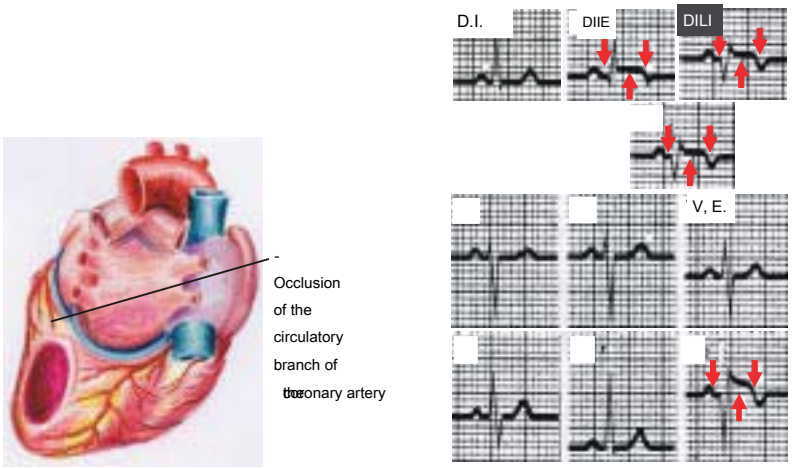


Fig. 44.16 Posterolateral infarction (in DI, DII, aVL and V).

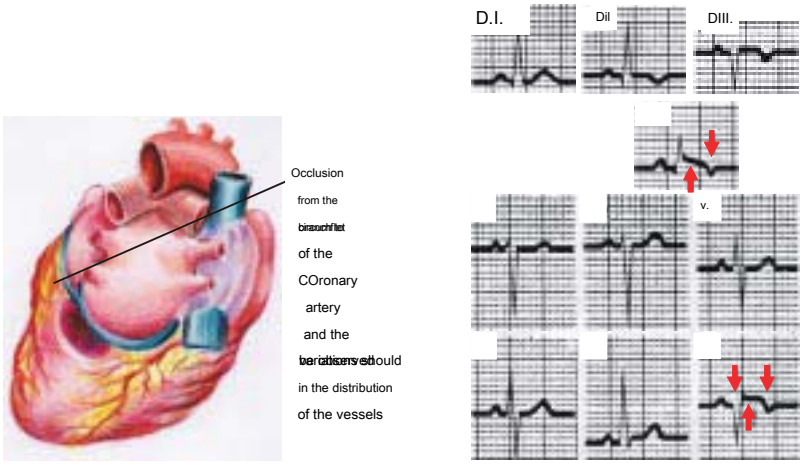


Fig. 44.17 Posterosuperior, posterobasal infarction (in aVF

Intramural infarctions. They are located in the middle third of the wall of the left ventricle.

The electrocardiographic criteria are those of ischemia and injury:

1. ST segment with positive displacement and deeply inverted T from V, to V,.

2. Absence of sign of necrosis (pathological Q wave).

Transmural infarcts. In the affected area, the lesion occupies the entire thickness of the wall of the left ventricle. The affected area would act as a kind of "electric window" that would allow the electrode facing it to record the QS complexes typical of the normal left cavity. The electrocardiographic criteria that characterize them are: QS complexes with negative T in the leads that explore the affected wall.

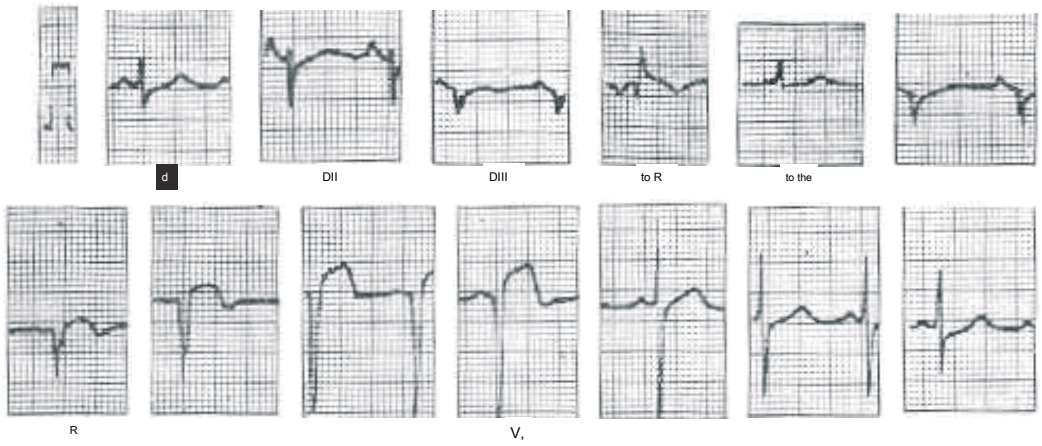


Fig. 44.18 Deep septal infarction.

Subepicardial infarctions. The electrocardiographic criteria are those characteristically described by Pardee, in which signs of injury appear (positively displaced ST), signs of ischemia (inverted and symmetrical T wave) and necrosis (pathological Q wave).

The evolutionary diagnosis determines the different stages through which the disease passes (see Fig. 44.9):

- Acute or early phase.
- Subacute or secondary phase.
-Chronic or late phase.

In the acute stage or early phase, the lesion predominates, characterized by marked displacement of the ST segment. The duration of this phase is hours, generally days, at most two weeks. The longer the ST segment remains displaced, the worse the prognosis will be. A displaced ST segment beyond the third week suggests a complication: aneurysm of the ventricular wall or extension of the infarct.

In the subacute stage or secondary phase, the cells most affected by the injury evolve towards definitive necrosis and the least affected will partially recover, persisting with a certain degree of ischemia. Consequently, this stage is characterized by the return of the ST segment to the isoelectric line, an increase in the pathological Q wave and the presence of the strongly inverted, symmetrical and pointed T wave; T wave inversion persists for four to six months, sometimes longer.

In the chronic stage or late phase, only the pathological Q wave persists, the ST segment is located on the isoelectric line and the T wave becomes isoelectric or even positive, generally remaining flattened. There should be no further changes and the route is stabilized.

Chronic coronary insufficiency

The fundamental lesion that causes it is coronary atheromatosis; The left ventricle and interventricular septum are preferentially affected in multiple areas.

of subendocardial fibrosis (microinfarcts). It is the well-known cardiosclerosis. It is observed in the elderly and is generally accompanied by heart failure and left ventricular hypertrophy.

The most frequent electrocardiographic criteria are:

1. Left axial deviation.
2. Flattened T waves with prolonged QT interval and associated with rhythm disorders, such as:
   a) Intense bradycardia (except if the patient is in heart failure).
   b) First degree atrioventricular block.
   c) Intraventricular conduction disorders, which give QRS complexes with low voltage gaps and prolonged duration.
   d) Frequent ventricular extrasystoles.

Due to their frequent presence in ischemic heart diseases, we will refer here to focal blogging, known in the medical literature by various names, the most correct and acceptable being peri-infarct block. They are related to a delay in the excitation wave (depolarization) at the level of the ventricular wall damaged mainly by an area of necrosis.

It includes both the so-called arborization or reticular blocks and the parietal or fibrillar blocks.

The electrocardiographic criteria will be given by increased QRS duration, up to 0.12 s or more.

OTHER ALTERATIONS OF ST T

Other ST and T wave alterations accompany other disorders, such as pericarditis, digitalis effect and electrolyte disorders, which, as they are accompanied by other manifestations, will be seen separately later.

RHYTHM ALTERATIONS ARRHYTHMIAS

A brief account of the formation and propagation of the electrical impulses which carry the contraction command to

nonspecific myocardial fibers, will allow a better understanding of cardiac arrhythmias.

The sinus or sinoatrial node of Keith and Flack, or more specifically its central cells, located in the right atrium near the mouth of the superior vena cava, governs the activity of the heart, which is why it is called the pacemaker or primary pacemaker. Its action is automatic with an average frequency of 70 discharges/ min. The heart rhythm governed by this node is called sinus rhythm.

The sinus electrical impulse is conducted by three intra-atrial pathways, also specific: the internodal bundles of James, which are directed towards the area known today as the junctional region, located in the lower part of the right atrium, near the interatrial septum, and more specifically in the atrioventricular node of Aschoff Tawara - the most differentiated portion of the aforementioned junctional region and endowed with automation-, with a frequency of approximately 50 discharges/min.

From the anterior internodal bundle, Bachmann's bundle emerges, which is directed towards the left atrium.

The stimulus that has crossed the region of the junction reaches directly to the bundle of His and from this it passes to the walls of the ventricles through a trifascicular system, whose new anatomohistological and functional conception we have already mentioned in Section I, Chapter 10.

The highly subdivided branches of this trifascicular system penetrate the thickness of the anterior papillary muscle of the right ventricle and the anterior and posterior papillary muscles of the left ventricle, constituting the terminal Purkinje network.

Under normal conditions, the sinus node is the one that originates the stimuli and thus begins the sinus rhythm. If for any reason the atrioventricular node takes control, a nodal or junctional rhythm is initiated.

Sometimes the stimuli arise below the A-V node, in the branches of the bundle of His, and then give rise to the idioventricular rhythm.

Arrhythmias can be classified into:

A. Arrhythmias due to disorders in the formation of the electrical impulse:

ì Sinus tachycardia
i Sinus bradycardia
Sinus arrhythmia
ormot pica Sinoatrial block Sinus arrest
1 Migratory pacemaker
; Extrasystoles
Paroxysmal tachycardias
eterot pica í Atrial fibrillation Flutter or flutter
i earphone
î Atrioventricular junction rhythms

B. Arrhythmias due to disorder in the conduction of the electrical impulse:

ì ìFirst grade
Atrioventricular iSecond grade:
and ular blocks í Mobitz Type I i
retarded Mobitz II Type
1 îThird grade

Branch blocks
Arborization blocks Olff-

Accelerated Parkinson syndrome hit

There are many other rhythm alterations, but we only point out the most significant ones and of them we will describe the most frequent ones in clinical practice.

ARRHYTHMIAS DUE TO DISORDER IN THE FORMATION OF THE ELECTRICAL IMPULSE

Arrhythmias due to a disorder in the formation of the electrical impulse are divided into two large groups: normotopic and heterotopic, depending on whether the alteration originates, respectively, in the sinus node or in an ectopic focus that has become more excitable.

Normotopic arrhythmias

Sinus tachycardia

The sinus stimulus is generated at an exaggerated frequency (between 100 and 150 beats/min). The conduction and propagation of the stimulus are carried out normally, so the morphology of the tracing waves is normal (fig 44.19).

The characteristic electrocardiographic manifestation is the shortening of the PT space, which shows the precipitation with which the stimulus is regenerated in the sinus node; Sometimes, the TP space is nonexistent, the P wave begins before the T of the preceding cycle has returned to the isoelectric line, and the P wave is recorded mounted on the T.

In sinus tachycardias, the ST segment may be slightly depressed, probably due to the appearance in said location, due to tachycardia, of the atrial repolarization wave or to relative ischemia, due to increased myocardial oxygen demand. The T wave may flatten and even persist for some time after the tachycardia disappears.

Semidiagnosis

1. Sinus tachycardias due to increased sympathetic tone: effort tachycardias and emotional tachycardias, hyperthyroidism. In some asthenic people, the mere act of getting up causes orthostatic tachycardia.

2. Toxic sinus tachycardias: due to atropine, which inhibits the vagus; by adrenaline, which stimulates the sympathetic; either

Fig. 44.19 Sinus tachycardia.

due to tea, coffee, nicotine, nitrites, etc., and febrile states of toxic infectious diseases.

3. In states of shock or hemorrhage.
4. In cases of organic heart diseases: cardiac rheumatism, endocarditis, pericarditis, valvular disease and heart failure.

Sinus bradycardia

It is caused by a decrease in sinus automatism, with the stimulus being repeated at longer time intervals than normal.

Electrocardiographic criteria (fig. 44.20)

1. Prolongation of the PT diastolic space.
2. Frequency between 40 and 60 beats/min.
3. The PR interval is lengthened, but usually does not exceed the maximum normal limit; of 0.20 s.
4. The QT interval is somewhat prolonged, but within normal limits according to the frequency.

Semidiagnosis

1. Constitutional bradycardia, hereditary in some families.
2. Athlete's bradycardia.
3. Bradycardias due to increased vagal tone, due to irritation of the vagus. Compression of the carotid sinus, Oculocardiac reflex, increased intracranial pressure (cerebral edema, meningitis, brain tumor). Also in infectious states (flu, other viral states, typhoid fever).
4. Toxic bradycardias. Digitalis impregnation or saturation, jaundice and quinidine overdose.
5. Metabolic bradycardias. In myxedema, beri-beri and atherosclerosis.

sinus arrhythmia

In this arrhythmia, the stimulus originates normally in the sinus node, but is not repeated at fixed intervals, but rather asynchronously, which have a margin of variability greater than 0. 12 s. The diastolic periods or TP space are of different duration.

There are three types of sinus arrhythmia: respiratory (Fig. 44.21), simple and bradycardia-tachycardia syndrome.

Respiratory arrhythmia is produced by variations in vagal tone that are conditioned by the influence of the respiratory act. The heartbeat rate accelerates during inspiration due to decreased vagal tone and is delayed with expiration. Apnea makes the respiratory arrhythmia disappear.

Simple arrhythmia is also conditioned by variations in vagal tone, but is independent of respiratory influence. The rhythm is delayed when vagal action predominates. It is distinguished from respiratory disease in that apnea does not make it disappear, but the injection of atropine does.

The bradycardia-tachycardia syndrome is also independent of respiratory influence and is conditioned by ischemia of the sinus node, due to sclerosis of its central artery and as an expression of atherosclerosis.

Fig. 44.20 Sinus bradycardia.

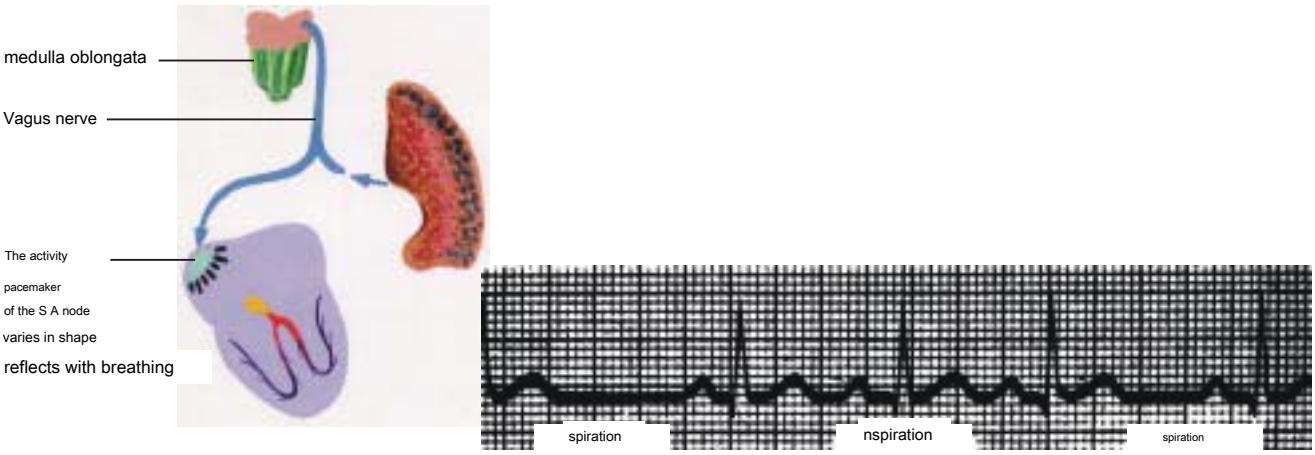


Fig. 44.21 Respiratory sinus arrhythmia.

coronary. It is also called sick sinus node or sinus node disease. It is expressed on the electrocardiogram by sinus bradycardia with sudden periods of sinus tachycardia, or vice versa, independent of respiration.

Semidiagnosis

Respiratory sinus arrhythmia is common and constitutes a normal phenomenon in vagotonic children. In adults it suggests an elevation of the diaphragm due to dyspeptic disorders, a post-infectious state or a senile heart.

The bradycardia-tachycardia syndrome is exclusive to senile hearts with atherosclerotic ischemic heart disease.

sinus arrest

It consists of the lack of generation of a stimulus within the sinus rhythm. It is attributed to a vagotonic reflex.

In the electrocardiogram it is characterized by the absence of a heartbeat, which causes a prolonged diastolic pause between two normal heartbeats. The duration of this pause is, generally, a little less than that corresponding to two normal cycles.

If sinus arrest is prolonged it can lead to death or the appearance of other types of arrhythmia (Fig. 44.22).

Migratory pacemaker

It consists of changing the starting site of the stimulus. This variation may be small and occur only within the same sinus node or neighboring areas of the atrium;
or greater, due to moving from the sinus node to the atrioventricular node.

In the first case, that is, when the variation is small and the change occurs within the same sinus node, or from it to nearby atrial areas, the tracing displays, in the same lead, P waves of variable shapes in the different complexes.

The P waves change direction little, they are always positive, since the vector resulting from atrial activation is always directed from above to below. PR intervals may vary slightly, but are always greater than 0.12 s.

In the second case, when the variation is greater and the change occurs from the sinus node to the atrioventricular node, the stimulus sometimes emerges through the sinus node and other times through the atrioventricular node, which gives rise to a heterotopic component in this type of arrhythmia (Fig. 44.23).

The electrocardiographic criteria will, therefore, be:

1. Negative P waves, especially in DII, DII and aVE, which will alternate with variable positive P waves.
2. The PR intervals of negative P waves will be shorter than 0. 12 s.

This arrhythmia in either of its two varieties is determined by the action of the vagus, so it may occur in subjects with alterations in vagal tone.

Eterotopic arrhythmias

Heterotopic arrhythmias can be passive and active.

Passive heterotopic arrhythmias are those that appear when the sinus node, for some reason, ceases to produce the stimulus, or the stimulus generated by it is blocked; As a consequence, a temporary cardiac arrest occurs, which is manifested in the electrocardiogram by a prolonged diastolic pause and which gives rise, under these conditions, to another stimulus-generating center becoming a pacemaker, and the arrhythmia is established as a physiological safety mechanism.

If this sinus arrest is momentary, a contraction called sporadic escape systole, or escape beat, occurs within a normotopic rhythm, if the ectopic center manifests only once. But if ma-

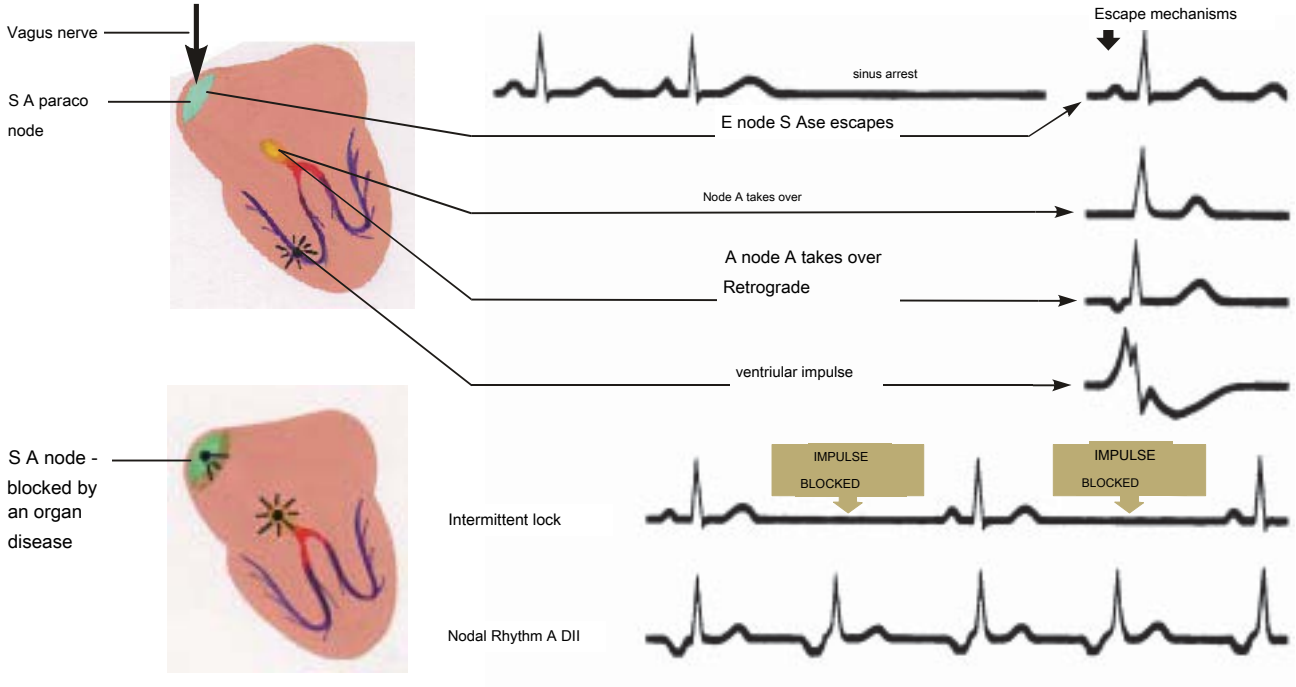


Fig. 44.22 Sinus arrest.

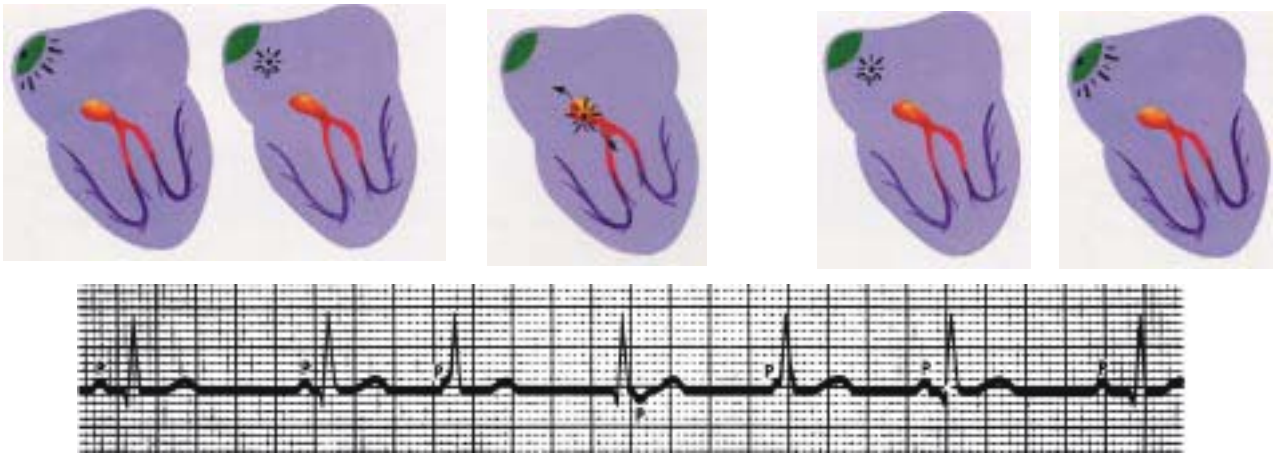


Fig. 44.23 Migratory pacemaker.

If the party is prolonged, the ectopic center takes over, and a replacement rhythm occurs.

These passive heterotopic arrhythmias are characterized by the delay with which contractions appear, not only the first beat, separated from the normal one that precedes it by a long diastolic pause, but also all the rest, as the frequency that generates stimuli from this ectopic, or secondary, center is much lower.

Active heterotopic arrhythmias are those that occur when a lower center experiences a state of abnormal excitability, which causes it to generate stimuli at a much faster frequency than that of the sinus node, in fact becoming, by this mechanism, a pacemaker. If it is limited to a single contraction, an extrasystole occurs, but if it is permanent, a paroxysmal tachycardia occurs. Heterotopic arrhythmias

Active contractions are characterized by the prematurity and rapidity with which contractions appear.

Morphology of complexes according to ectopia

The form of the complexes, whether an active or passive heterotopic rhythm, will depend on the place where the stimulus originates.

The bundle of His constitutes the dividing zone that allows the separation of ectopias into two large groups, which will show different QRS complexes. Stimuli originating above the bundle of His or in it give rise to the appearance of QRS complexes of normal morphology, as the ventricles depolarize following the normal pathway: they are the so-called supraventricular type complexes. All the stimuli that originate from

Below the division of the His bundle, they give rise to wide and deformed QRS complexes, due to greater slowing of conduction, and usually with opposing T waves.

When the ventricular muscles depolarize in whole or in part through abnormal means, the so-called aberrant QRS of the ventricular type is produced.

When the stimuli originate in the atrium, outside the sinus node, in a focus located in the upper part of the atrium, the P waves are hardly deformed and the PR interval is little altered in its duration. As the stimulus originates in lower areas of the atrium, the P wave becomes inverted and the PR becomes shorter. If the stimuli originate in the junctional region, in its upper part, a negative P wave is produced and the PR is shortened even more and if the stimulus originates in the middle part of the junctional region, it reaches the atria and ventricles at the same time and the P wave coincides with the QRS complex, so it is not seen in the tracing.

If the stimulus originates in the lower portion of the junctional region, it reaches the ventricles first than the atria, producing a QRS complex followed by an inverted P wave over the ST segment. If it originates in the bundle of His, the QRS complex is slightly deformed, but there is no P wave present, since there is normally a physiological block from below to above in the atrioventricular node.

When it originates in the wall of the ventricle, the propagation is slow, as it travels in a tissue not suitable for conduction, producing wide, slowed, indented QRS, generally of high voltage, the so-called aberrant QRS and an opposing T wave. There is no P wave.

Among the heterotopic arrhythmias, the most important are:

xtrasystoles

They are premature contractions originating from an ectopic focus. They can be: atrial, nodal and ventricular.

Atrial extrasystoles are premature contractions originating in the atrium; They present the following electrocardiographic criteria (fig. 44.24):

1. Presence of P wave, positive if they originate in the upper portion of the atrium, and negative if they originate in the lower portion.
2. The PR interval is 0.12 s or greater.
3. The QRS complex is of the supraventricular type, without aberrant conduction.
4. There is no complete compensatory pause (see below).

Nodal extrasystoles are premature contractions originating in the atrioventricular node; They present the following electrocardiographic criteria:

1. The P wave may or may not be present. When it is, it is retrograde, negative in DII, DIII and aVE.
2. The PR interval, when it exists, is less than 0.12 s.
3. The QRS complex is of the supraventricular type.
4. In the vast majority of cases there is a complete compensatory pause (see below).

Depending on the place in this region where they originate, they can be divided into: supranodal, mesonodal and infranodal extrasystoles, whose characteristics correspond to the morphological description of the complexes according to the ectopia already described (fig. 44.25).

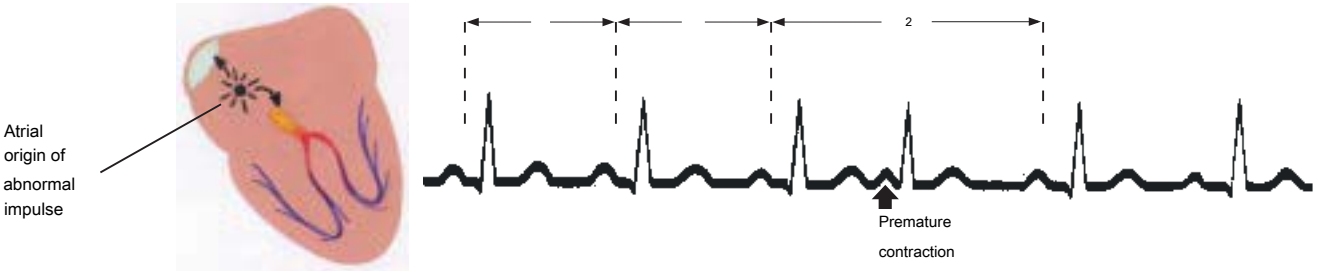
Ventricular extrasystoles are premature contractions originating in the ventricular walls or in one of the branches of the bundle of His. They are the most frequent, they present with the following electrocardiographic criteria (fig. 44.26):

1. They are not usually preceded by P waves. There are occasions when it is possible to observe the normal P wave over extrasystoles and other times, very rarely, an inverted P wave.
2. There is, therefore, no PR interval.
3. Aberrant ventricular-type QRS complexes.
4. Opponent T waves and generally of great voltage.
5. They have a complete compensatory pause.

The lengthened diastolic space that exists after extrasystole is called compensatory pause. It can be complete or incomplete.

When the distance between the P wave of the QRS complex, which precedes the extrasystole, and the P wave of the QRS complex, which follows it, is equal to the distance between

Fig. 44.24 Atrial extrasystoles.



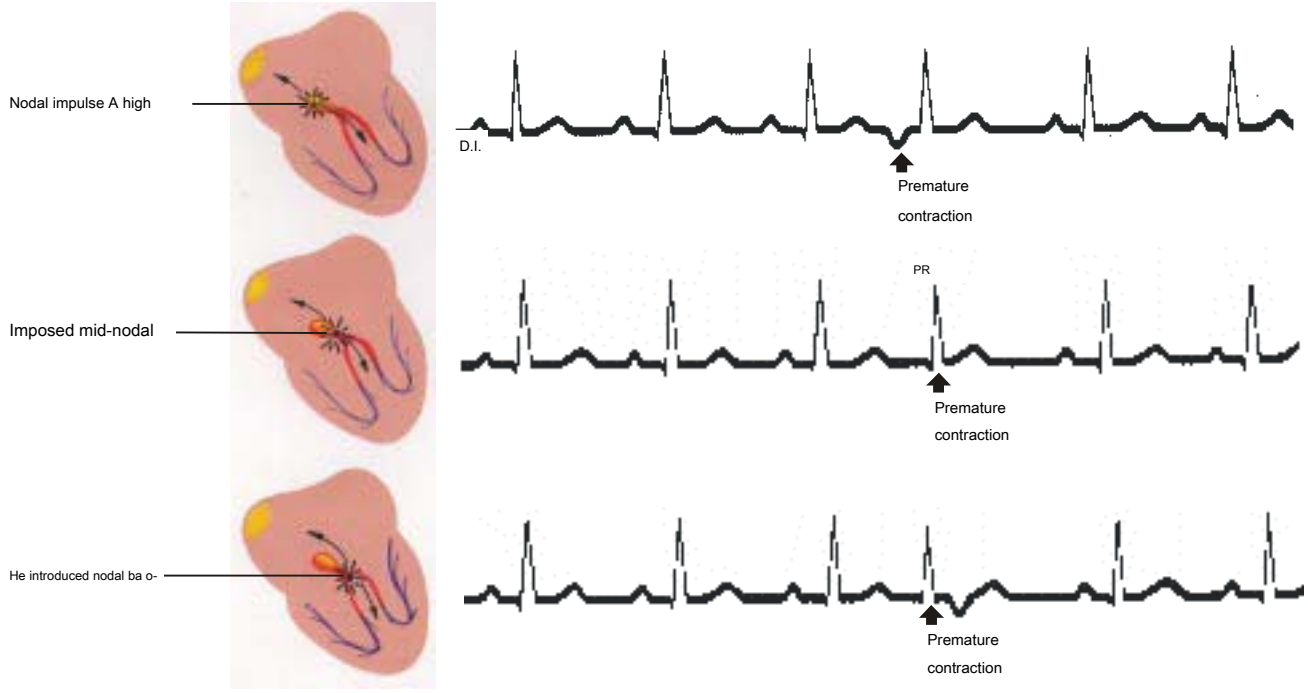


Fig. 44.25 Nodal extrasystoles.

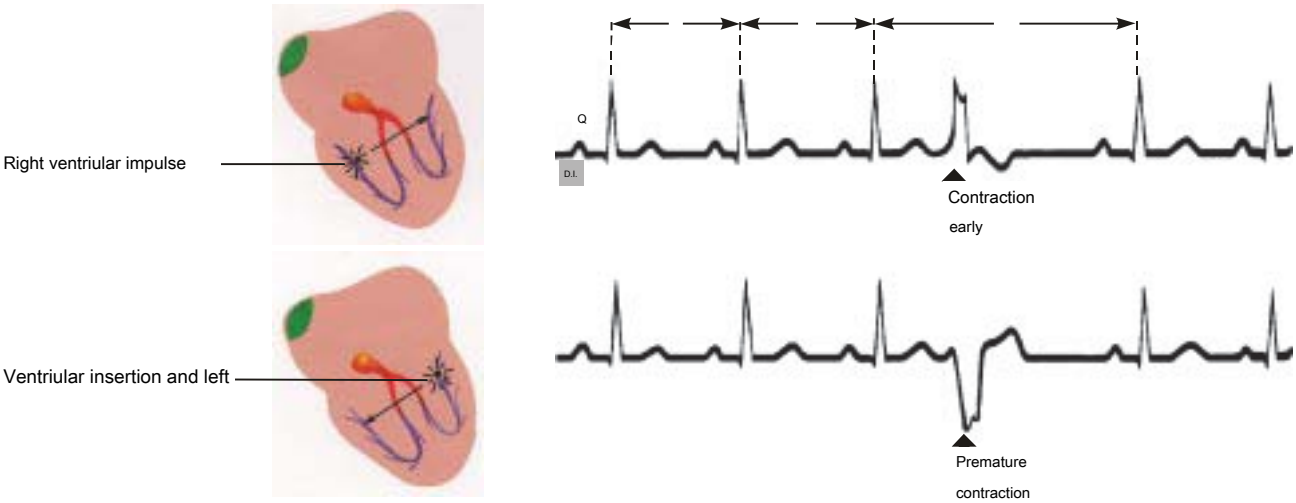


Fig. 44.26 Ventricular extrasystoles.

two normal cycles, it is said that there is a complete thinking pause. Ventricular extrasystoles have a complete compensatory pause.

When the distance between the P wave of the QRS complex, which precedes the extrasystole, and the P wave of the QRS complex, which follows it, is less than twice the distance between two normal cycles, there is said to be an incomplete compensatory pause. Atrial extrasystoles have an incomplete compensatory pause.

Paroxysmal tachycardias

They can be considered, as a general rule, as the uninterrupted appearance of extrasystoles. They constitute a active heterotopic rhythm. The heart rate ranges in these arrhythmias between 150, 200 or more beats/min, although there may be exceptions.

They are classified into paroxysmal supraventricular tachycardias (atrial and nodal) and paroxysmal ventricular tachycardias.

Paroxysmal supraventricular tachycardias

In this type of arrhythmia, an uninterrupted succession of extrasystoles occurs, either atrial or nodal, adopting the name supraventricular, since it is very difficult to differentiate in this type of tachycardia, the auricular ones from the nodal ones, and much less so.

us the exact site of the origin of the stimulus within the atrium or the region of the atrioventricular node. Since both tachycardias respond to the same treatment, the important thing is to be able to diagnose that the paroxysmal tachycardia is supraventricular, without trying - because it is difficult - to specify whether it is atrial or nodal.

Electrocardiographic criteria

1. Its frequency ranges between 150 to 200 beats/min, generally.
2. They have a P wave, but it can rarely be identified.
3. Supraventricular type QRS complex with normal morphology and duration, although sometimes it may be somewhat deformed due to certain aberrant conduction, making diagnosis difficult in these cases.
4. Slight negative ST segment elevation, sometimes with a negative T wave, due to changes secondary to tachycardia or primary changes due to coronary insufficiency.

Other clinical criteria that help with the electrocardiographic interpretation of paroxysmal tachycardias are:

1. They appear in the form of sporadic attacks, with a sudden start and end.
2. They obey the law of all or nothing, that is, when a vagal excitation maneuver occurs they disappear completely or are maintained (fig. 44.27).

Paroxysmal ventricular tachycardias

They consist of an uninterrupted succession of ventricular extrasystoles. They do not respond to the all-or-nothing law, nor do they yield to vagal stimulation maneuvers.

Electrocardiographic criteria (fig. 44.28)

1. The rate is high, generally greater than 200 beats/min.
2. P waves occur with normal frequency and morphology, but usually cannot be identified by coincide with the aberrant QRS complex. If they can be identified, they are independent of QRS, and can precede it, follow it or coincide with it, as we have just pointed out, contributing to deforming it even more.
3. Aberrant ventricular QRS complexes, wide, deformed, with indentations, with opposing T waves.

This tachycardia can lead to ventricular fibrillation, which is a premortem state (Fig. 44.29).

Semidiagnosis

Paroxysmal ventricular tachycardias occur in patients with severe organic heart disease, with poor general condition, accompanied by serious complications (acute pulmonary edema).

Occasionally, this arrhythmia may appear due to the administration of digitalis.

atrial fibrillation

It consists of a rapid and irregular parcel depolarization of the atria, which results in the production of a series of small and frequent contractions, without there being an effective atrial systole, from a mechanical point of view. The frequency of these small contractions is greater than 400 impulses/min and of every three or four of these impulses, the atrioventricular node is only capable of responding to one, which causes the variable ventricular rate, generally greater than 100 beats/min, but may also be lower.

Electrocardiographic criteria

1. Absence of P waves; A series of light and irregular undulations of the baseline are observed on the isoelectric line, representing the small electromotive force produced by the parcel depolarization of the atria. They are the so-called f waves, which are best appreciated in leads DII, V, and V,.
2. Asynchronous registration of QRS complexes of the supraventricular type, but whose configuration can

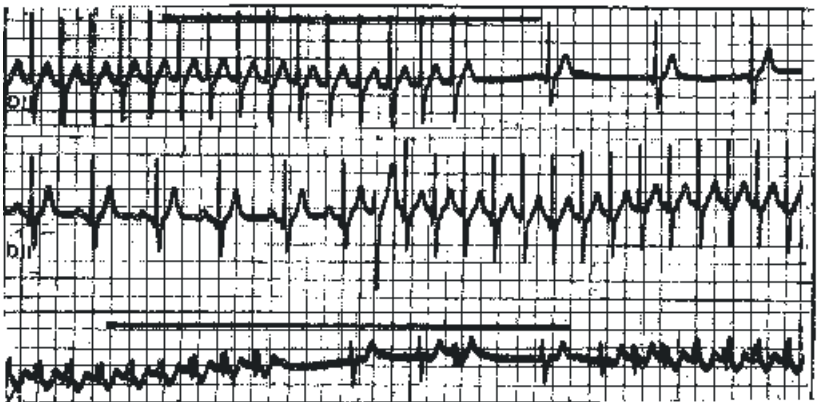


Fig. 44.27 Paroxysmal supraventricular tachycardia (compression of the carotid sinus).

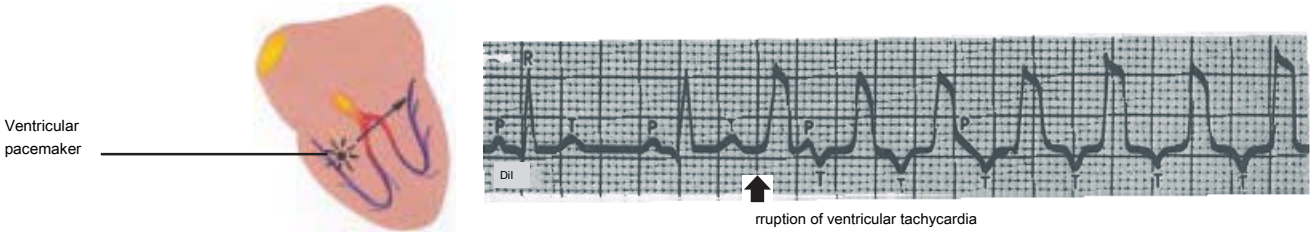


Fig. 44.28 Paroxysmal ventricular tachycardia.

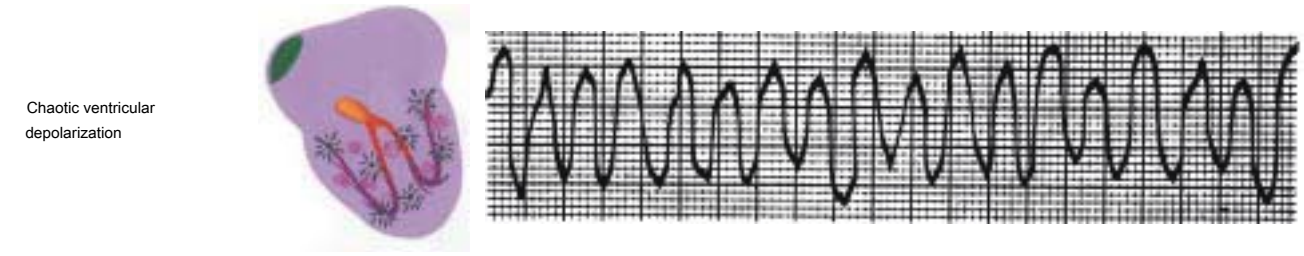


Fig. 44.29 Ventricular fibrillation.

vary slightly, depending on the aberration of the greater or lesser refractory period in which the ventricular musculature finds itself when it enters the stimulus from the atrium. Atrial fibrillation can be fast or slow, depending on the ventricular rate (Fig. 44.30).

Semidiagnosis

1. Atherosclerotic heart disease.
2. Rheumatic heart disease.
3. Hypertensive heart disease.
4. Cardiothyrotoxicosis.
5. Congestive heart failure.
6. In apparently healthy subjects (usually paroxysmal) without a history of heart disease.

atrial lutter or flutter

In all probability, it is due to a circular movement of the stimulus that runs around the orifices of the vena cava, or to the existence of a high-frequency atrial ectopic pacemaker.

The ventricles can respond to each atrial stimulus with a 1:1 rhythm, but it is more common for them to do so every two, three or four stimuli, thus obtaining 2:1 rhythms; 3:1; 4:1, etc.

Electrocardiographic criteria

1. P waves with an absolutely regular rhythm, whose ascending branches are steep and the descending branches are gradual; They occur at a high frequency, 200 to 400/min, which means that there is practically no isoelectric interval between these waves, called EĘ waves, which give the baseline a characteristic scalloped, sawtooth appearance.
2. QRS complexes of the supraventricular type with a regular frequency, generally between 70 and 100 beats/min, although it can become irregular, due to variable atrioventricular block (fig. 44.31).

Semidiagnosis

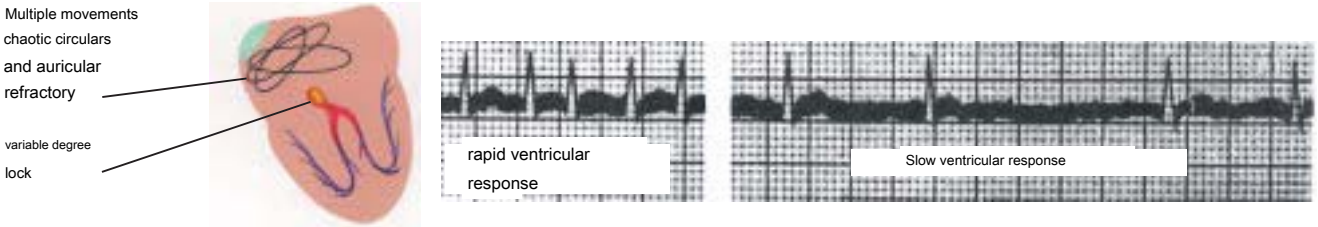
1. Pulmonary artery disease.
2. Cor pulmonale.
3. Rheumatic valve disease.
4. Rarely induced by digitalis.

Atrioventricular junction rhythms

Also known as nodal rhythms.

The name junctional rhythms is the most appropriate because it has been difficult to demonstrate convincingly that there is heart rate regulation activity in all regions of the node and also because the activity

Fig. 44.30 Atrial fibrillation.



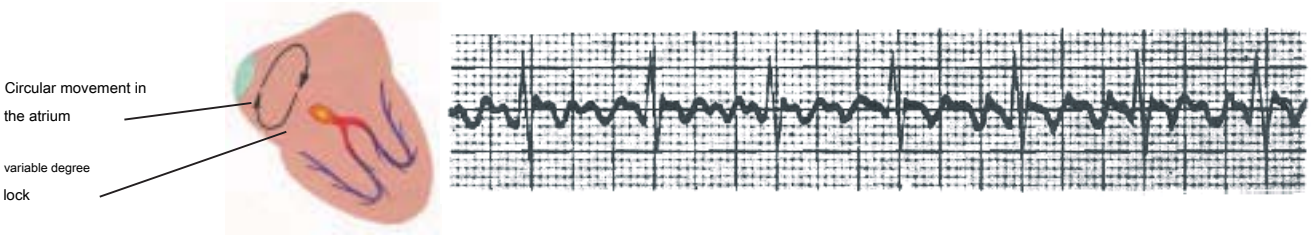


Fig. 44.31 | e earpiece.

Pacemaker activity in other areas (coronary sinus, left atrium and bundle of His) can produce electrocardiographic tracings with characteristics similar to those of the A-V node rhythms.

They are primarily escape rhythms with a frequency, usually between 40 and 70 beats/min.

They manifest in cases of significant decrease in sinus rate, sinoatrial block and atrioventricular block.

Electrocardiographic signs (Fig. 44.32)

1. QRS complexes similar to those of sinus rhythm.
2. Other electrocardiographic criteria, such as morphological characteristics of the P wave and its relationship with the QRS complex, have already been described in the morphology of the complexes according to ectopia.

ARRHYTHMIAS DUE TO DISORDER IN THE CONDUCTION OF THE ELECTRICAL IMPULSE

Delayed arrhythmias

Of these arrhythmias, bundle branch blocks and arborization blocks have already been studied previously; here we only have to refer to atrioventricular blocks.

atrioventricular blocks

In this arrhythmia, the conduction disorder lies in the fact that the passage of the excitation wave is difficult or prevented at the level of the atrioventricular node.

They are classified into three types: first, second and third grades.

First degree atrioventricular block

Electrocardiographic criteria (Fig. 44.33) 1. PR interval with a duration of 0.20 or greater.

It must be remembered that the PR interval varies greatly with heart rate, lengthening as the rate decreases, but generally a PR greater than 0.20 s is pathological, no matter how low the frequency. In cases of tachycardia, reference tables should be consulted.

Semidiagnosis

1. Functional causes: in athletes.
2. Organic causes: degenerative conditions of the myocardium, acute articular rheumatism, diphtheria.
3. Toxic causes: digitalis poisoning.

Second degree atrioventricular block

It has been classified into two types: Mobitz I and Mobitz I.

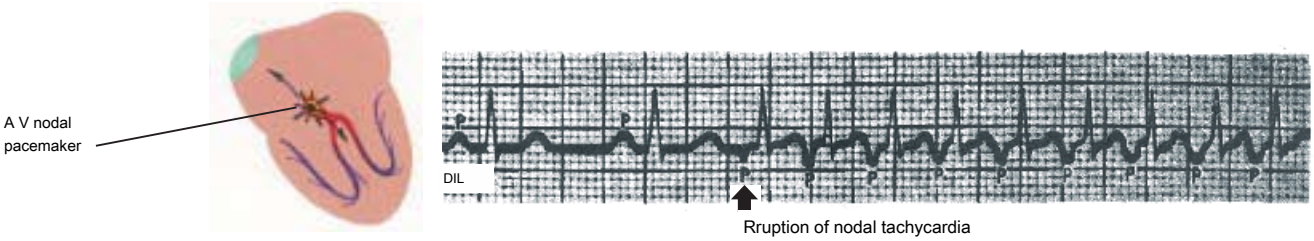
Second-degree atrioventricular block type Mobitz I is characterized on the electrocardiogram by (see Fig. 44.33) :

1. Progressive increase in duration of the PR interval (Wenckebach-Luciani phenomenon), which precedes a non-conducted sinus stimulus, that is, a P that is not followed by a QRS complex.
2. After n0 a P wave is conducted, the next PR interval is shorter, equal to the first of the previous cycle.

Second-degree atrioventricular block type Mobitz II is characterized on the electrocardiogram by (Fig. 44.34):

1. Constant PR intervals, before a P wave unexpectedly fails to conduct. Failed beats can occur in a regular manner, expressed in broken form, where the numerator indicates the number of waves P and the denominator the number of QRS complexes; example: out of every three impulses

Fig. 44.32 Tachycardic nodal rhythm.



Rruption of nodal tachycardia

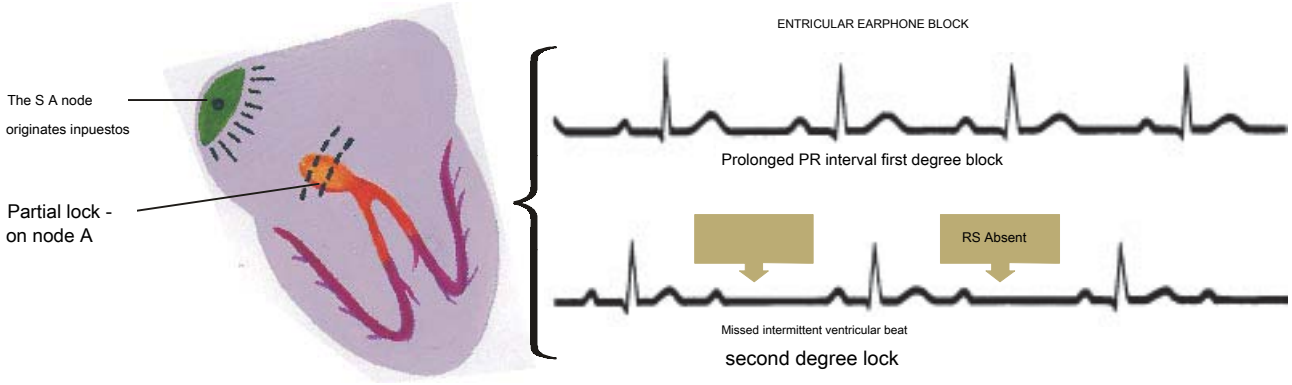


Fig. 44.33 First degree atrioventricular block. Second degree atrioventricular block. Mobitz I type.

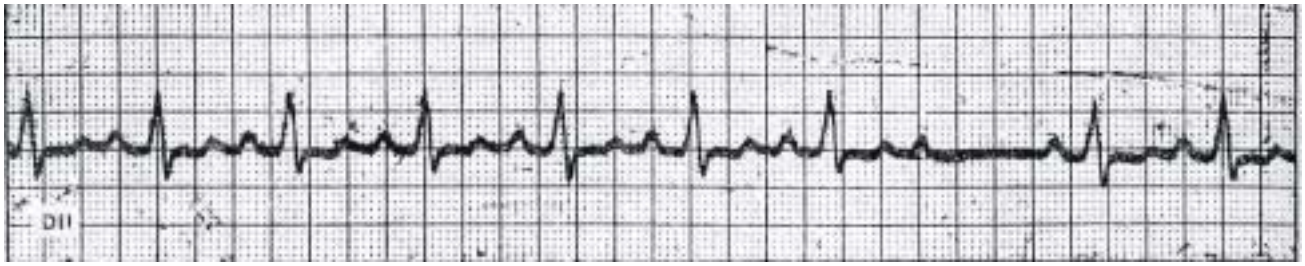


Fig. 44.34 Second degree atrioventricular block. Type Mobitz I|.

Only one of your headphones leads to the ventricle (3:1);
out of four, three do not lead to the ventricle (4:1), etc.

Third Grade Atrioventricular Blogging (Complete)

The stimulus originating in the atrium cannot cross the atrioventricular node and the ventricles have to beat with their own rhythm, establishing a passive rhythm, of the junctional region or indioventricular.

Electrocardiographic criteria (fig. 44.35)

1. The P-P distances are equal to each other and different from those of the QRS complexes, which in turn have the same distance from each other.
2. The atrial rate is much higher, approximately double the ventricular rate, which varies between 32 and 55/min and depends on the site of the distal subsidiary pacemaker, the lower the rate is.

minor.

3. There is, therefore, no PR interval, that is, a relationship between the P waves and the QRS complex; the P wave can fall before, after or coincide with the QRS complex and give the impression when observing the tracing of a very variable PR.
4. The morphology of the QRS complex will vary with the site of implantation of the ectopic focus.

In cases of complete atrioventricular block, with a very slow ventricular rate, there may be

There is a clinical picture characterized by a slow pulse of 30 to 40 beats/min with frequent convulsive seizures and loss of consciousness, these are called seizures or Stokes-Adams syndrome. Considerably decreased circulatory output is the cause of transient cerebral ischemia and syncope.

Semidiagnosis

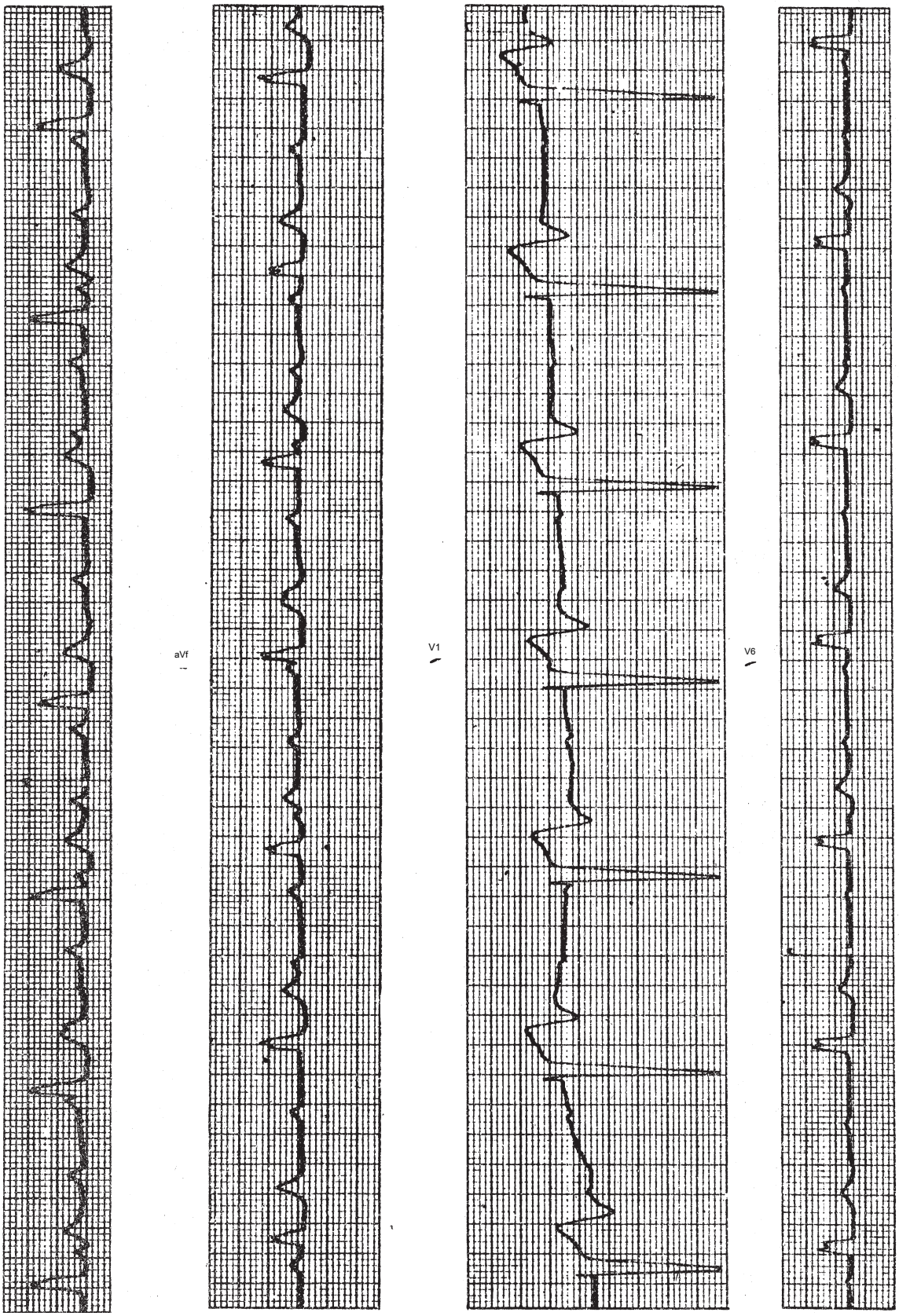
These rhythm disorders (atrioventricular blocks) are observed in patients with a diffuse degenerative process of the myocardium.

Accelerated arrhythmias

In this type of arrhythmias due to a disorder in the conduction of the electrical impulse we will refer to the Wolff-Parkinson-White (W-P-W) syndrome or pre-excitation.
The sinus stimulus reaches one ventricle through an anomalous pathway, through which the stimulus spreads quickly to the other ventricle, as it does not have to cross the atrioventricular node, which delays it due to its long refractory period. Once the stimulus reaches the ventricle quickly, it spreads slowly, especially in the first moments, because it invades an area devoid of specific tissue for conduction.

Electrocardiographic criteria

1. Short PR, lasting less than 0.12 s, there is practically no PR segment, observing the P wave followed by the QRS complex (fig. 44.36).


aVf
V1
V6

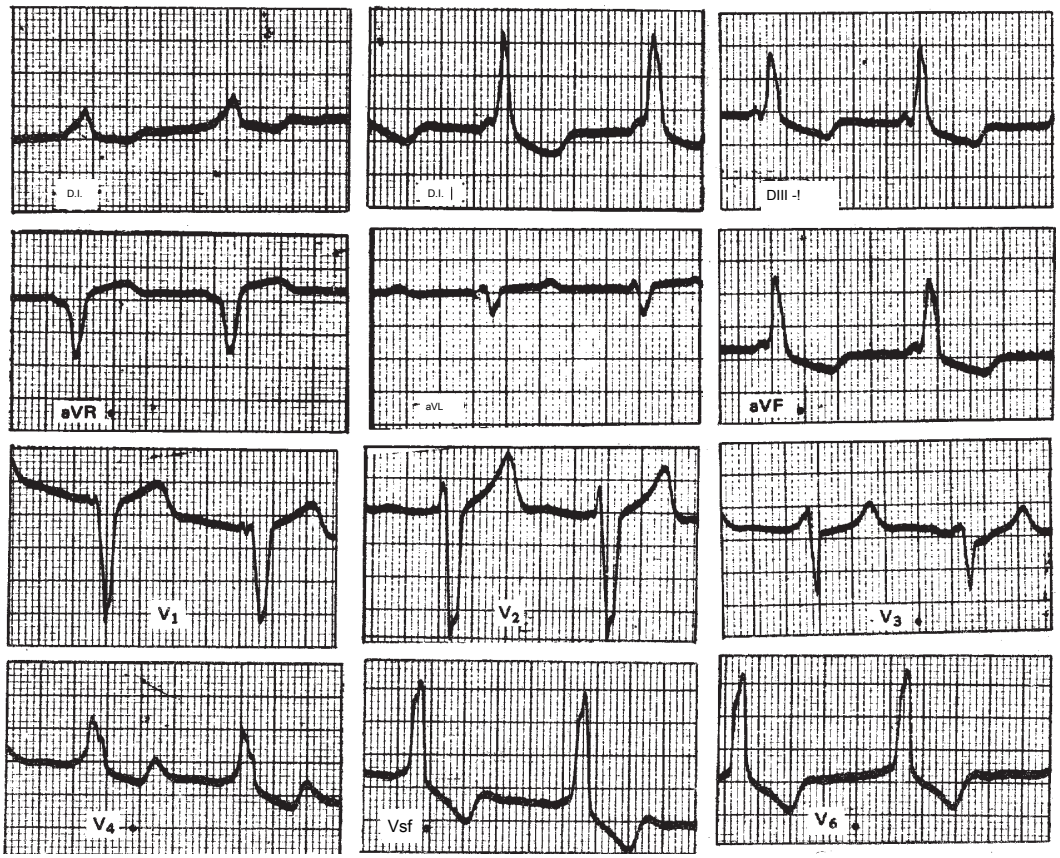


Fig. 44.36 Syndrome olff-Parkinson- hite.

2. QRS complexes of prolonged duration, 0.12 s or greater. Presence of delta wave: slowing or rounded notch at the beginning of QRS (fig. 44.37).

There are two varieties of W-P-W syndrome:

1. Type A, R waves are abnormally high in all precordial leads.

2. Type B, the most frequent, with the ORS of leads on the right precordium predominantly negative.

Semidiagnosis

1. Common in young people who suffer from attacks of ectopic tachycardia.

2. Ebstein's anomaly.

3. Cardiosclerosis.

4. In some cases of endocardial fibroelastosis.

CHANGES IN OTHER FREQUENT PATHOLOGICAL ENTITIES

In this section we are going to briefly refer to the electrocardiographic alterations observed in pericarditis, acute cor pulmonale, chronic cor pulmonale, alterations produced by digitalis and alterations produced by electrolyte disorders.

PERICARDITIS

The electrocardiographic manifestations of pericarditis do not depend directly on the inflamed serosa but rather on the concomitant alterations of the epicardial surface, which is affected by extension of the pericardial inflammatory process.

Electrocardiographic criteria

The electrocardiographic manifestations are the same as those of ischemia:

1. Positive ST segment elevation with inverted T waves.

2. The QRS complex does not show alterations. There is never a pathological Q wave.

3. The alterations appear at any point on the epicardial surface explored.

One of the most important characteristics consists of the rapid evolution of these signs, which schematically go through two phases:

Acute diffuse pericarditis phase

1. Positive shift of the ST segment, in all leads, being less noticeable in lead DIII. This positive ST displacement is of superior concavity.

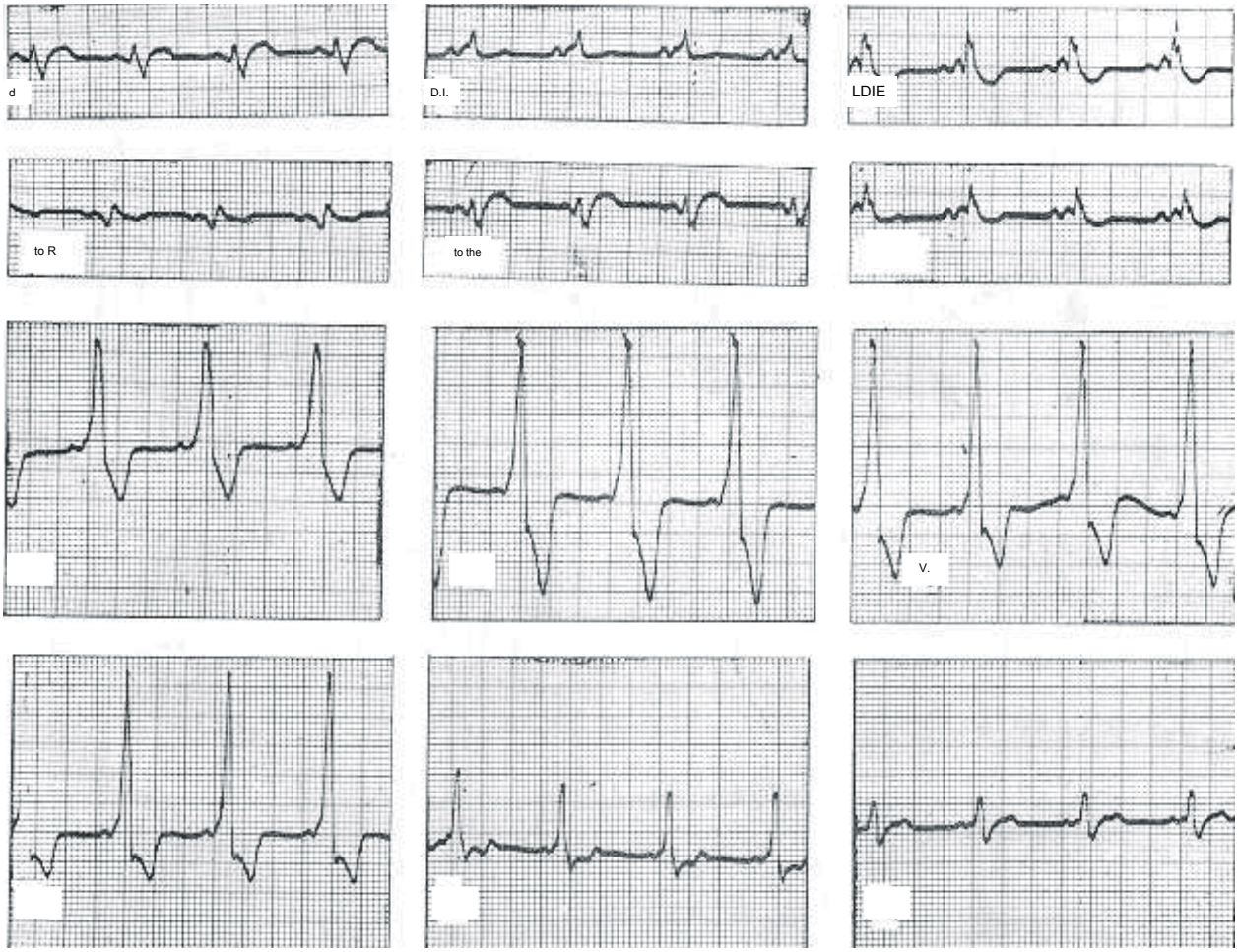


Fig. 44.37 Olff-Parkinson-hite syndrome (other modality).

2. The T wave shows few alterations in this phase, but can sometimes adopt an abnormally high and symmetrical morphology.
3. Usually there are no voltage alterations of the QRS complex.

4. These manifestations are short-lived: hours or days.

Subacute pericarditis phase

1. The ST segment gradually returns to the isoelectric line.

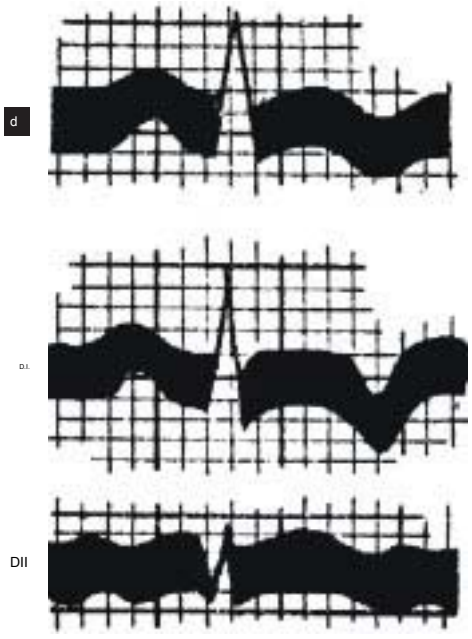
2. The T wave becomes depressed, flattens and becomes negative. This negative T wave appears in all leads that scan the epicardial surface of the heart. The T waves return to normal with the regression of the process.

Chronic constrictive pericarditis

Electrocardiographic criteria (fig 44 38)

1. Low voltage of the QRS complex.
2. Flattened or inverted T waves.
3. P waves of normal and even high voltage.

Fig. 44.38 Chronic constrictive pericarditis.



ACUTE COR PULMONALE

It is caused by sudden hypertension of the right ventricular chamber that dilates.

Electrocardiographic criteria

In standard leads (fig. 44.39):

1.S deep in ID, with negative ST segment.

2. Deep Q wave in DIII, sometimes accompanied by a negative T wave.

3. Absence of Q wave in aVF, thus allowing the differentiation of acute cor pulmonale from posterior wall cardiac

In precordial leads:

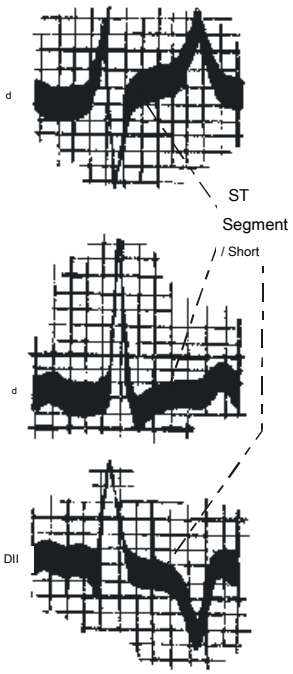
1. Inverted T wave in V,, V,, V, and in VE (special over the sternum and to the right).

2. Positive ST segment elevation, sometimes in the same leads.

3. QRS complexes remain unchanged, generally without deep Q wave.

If the patient survives, all these electrocardiographic signs disappear quickly in a few hours or days.

Fig. 44.39 Acute cor pulmonale.



CHRONIC COR PULMONALE

It originates as a consequence of hypertension maintained chronically for years at the level of the pulmonary

Electrocardiographic criteria

1. Tall and pointed P waves (pulmonary P) in DII, DIII and aVE one of the most characteristic signs.

2. Heart in vertical electrical position and axial QRS deviation to the right.

3. The complexes are usually of small voltage.
especially in limb derivations.

4. S wave important in DI.

5. In precordial leads. Deep S wave from V, to V, with flattened or inverted T waves.

6. Atrial fibrillation may be associated.

CHANGES PRODUCED BY DIGITAL

They are observed when administered in large doses or to hypersensitive individuals, or those suffering from myocarditis or cardiosclerosis.

Electrocardiographic criteria (fig 44 40)

1. Negative ST segment elevation with positive T waves: the ST segment adopts a characteristic "bucket" arrangement quite frequently, especially in DI, aVL, V,, V, and Vg

2. Shortening of the QT interval.

3. Signs of increased toxicity may appear; ventricular extrasystoles, with a serious prognosis if they are very numerous and multifocal; marked lengthening of the PR interval, with the appearance of first and second degree atrioventricular blocks; third degree or complete atrioventricular block; crisis of paroxysmal ventricular tachycardia and ventricular fibrillation.

ALTERATIONS PRODUCED BY ELECTROLYTIC DISORDERS

We will refer to the most frequent in medical practice: hypokalemia and hyperkalemia.

hypokalemia

It may occur as a consequence of renal or extrarenal losses.

Electrocardiographic criteria (fig 44 41)

1. Depression (negative elevation) of the ST segment.

2. Flattened T waves, which become progressively smaller in severe hypokalemia.

3. Elevated WaveU.

4. Serious arrhythmias in digitalized patients as a consequence of sudden hypokalemia.

Semidiagnosis

1. Kidney losses: due to diuretics (due to prolonged administration or overdose) or due to chronic conditions.

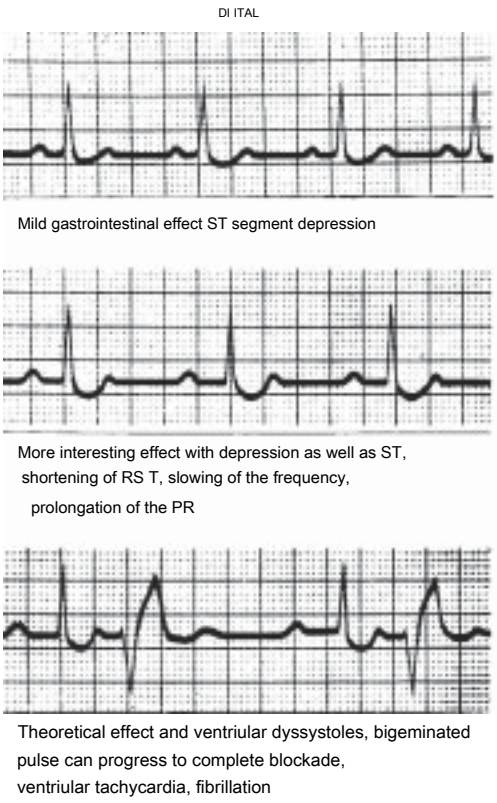

DI ITAL
Mild gastrointestinal effect ST segment depression
More interesting effect with depression as well as ST,
shortening of RS T, slowing of the frequency,
prolongation of the PR
Theoretical effect and ventriular dyssystoles, bigeminated
pulse can progress to complete blockade,
ventriular tachycardia, fibrillation

Fig. 44.40 Digital electrocardiographic alterations.

nal acidosis (renal acidosis of tubular origin and diuretic phase of tubular necrosis).

2. Extrarenal losses: vomiting, diarrhea, gastric drainage.

3. Treatment with adrenal steroids.

4. Primary aldosteronism.

5. Cushing's syndrome.

iperkalemia iperkalemia

It is frequently due to defective excretion of potassium by the kidney.

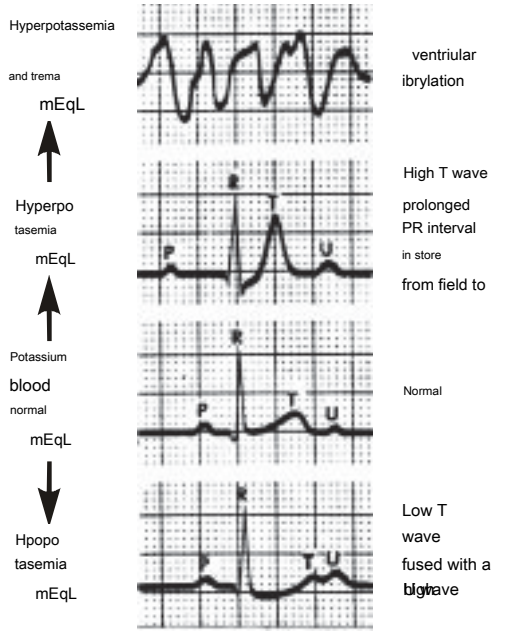


Fig. 44.41 Ipokalemia and hyperkalemia.

Electrocardiographic criteria (see fig 44 41)

1. “Tent-shaped high T waves.”

2. P waves with decreased amplitude, later atrial asystole.

3. Intraventricular block, with widened QRS complexes. In advanced cases ventricular arrest.

4. Electrocardiographic manifestations appear when the potassium concentration reaches 7-8 mEq/L.

Semidiagnosis

1. Primary kidney disease; acute tubular necrosis da and chronic kidney failure.

2. Adrenal steroid insufficiency: Addison's disease.

3. Use of aldosterone antagonists (spirolactones).

45

As we have done in the systems studied in previous chapters, in this chapter we will describe the main syndromes that can be observed in the cardiovascular system:

- Shock or shock syndrome.
- Heart failure syndrome.
- Coronary insufficiency syndrome.
- High blood pressure syndrome.
- Valve syndromes.
- Pericardial syndrome.
- Cardiac tamponade syndrome.
- Venous hypertension syndrome.

S 0C 0C0 UE SYNDROME

CONCEPT

Acute and complex clinical syndrome in which there is a severe and generalized decrease in nutritive blood flow to the tissues. If this tissue hypoperfusion is prolonged it leads to an irreversible deficiency in cellular function.

CLASSIFICATION

1. Hypovolemic shock.
2. Cardiogenic shock.
3. Distributive shock.
4. Vascular obstructive shock.

Hypovolemic shock is due to a decrease in actual circulating blood volume, as seen in the loss of blood, plasma, or water and salt or in the internal sequestration of these fluids in a hollow viscus or body cavity.

Cardiogenic shock is due to impaired pump function of the heart (pump failure), as seen in acute myocardial infarction or severe cardiac arrhythmias.

Distributive shock is due to vasomotor and microcirculation dysfunction from poor intravascular volume distribution related to excessive regional vasodilation, as seen in

severe localized or generalized sepsis or in significant spinal trauma or also in overdose of narcotic anesthetics.

Vascular obstructive shock is due to extracardiac mechanical obstruction of blood flow, as observed in obstruction to venous return (venas cava) or obstruction to ventricular ejection.

As the shock goes through stages from less to greater severity, it can also be classified as reversible or irreversible, which includes the stages of compensated or decompensated shock.

Compensated shock corresponds to an early stage of shock, with symptoms and signs that are not very apparent. In this case, blood pressure is maintained or drops slightly.

Decompensated shock is a more advanced stage, here the compensatory mechanisms that were put into operation during the previous stage to preserve the irrigation of vital organs are already insufficient and result in a greater deterioration of the irrigation of the main organs. At this stage it is still possible to reverse the situation if consistent measures are applied.

In irreversible shock, the excessive and prolonged reduction in tissue flow worsens the hemodynamic situation to extremes incompatible with life, and gives rise to the so-called simple organ failure.

SYNDROMOGENESIS PATHOPHYSIOLOGY

A common alteration to the different types of shock is the insufficiency of the circulatory system to maintain blood supply to organs and tissues and cellular functions.

To study the pathophysiology of shock, the structure and function of the circulatory system must be taken into account.

The three fundamental components of the system are:

1. The cardiac pump, contractile force of circulation.
2. Blood volume, the determining factor of preload.
3. The vascular system:
   a) Arteries.
   b) Veins.
   c) Microcirculation, allows the exchange of fluids and metabolites between the extra and intracellular compartments.

The basic functions of circulation are the supply of oxygen and essential nutrients to peripheral tissues and the removal of metabolic wastes from those tissues.

The irrigation of any organ is based on systemic arterial pressure (force that drives blood through of the organs), the resistance offered by the vessels of that organ and the permeability of the nutritional capillaries within it.

The determinant of substrate and metabolite exchange in tissues is microcirculation. The common denominator of all forms of shock is microcirculatory insufficiency, which can have a wide variety of causes. It is convenient to consider the pathophysiology taking into account the triggering causes, the effects of the shock process on the target organs and the vicious circles that contribute to maintaining and aggravating the syndrome.

Hemorrhages, acute myocardial infarction, severe sepsis, anaphylaxis, spinal cord injuries and anesthetics are some causes of shock, and although they are apparently unrelated, they all affect microcirculation;
Its most common impact is to produce a reduction in capillary perfusion that deprives cells of O and other necessary elements, allowing the accumulation of waste products that together determine membrane dysfunction.

SYNDROMOGRAPHY OR POSITIVE DIAGNOSIS

Clinical picture

The clinical picture of shock is somewhat variable because the event can range from mild to severe, from sudden onset to gradual and from short duration (minutes) to prolonged (days).

The presenting signs and symptoms of shock, which represent the manifestations of multisystem hypoperfusion and dysfunction, multiple organ dysfunction and failure, disseminated cellular injury and death, and body responses to this acute situation.

nazante

Although the different specific causes of shock produce very varied clinical and hemodynamic manifestations. In general the following data appears:

- Mental status: anxiety at first, later confusion, agitation, lethargy, drowsiness and coma in prolonged to severe stages.
- Blood pressure: it is normal at the beginning of the shock because the compensatory mechanisms are effective; Later, hypotension is established with figures less than 90 mm Hg. La hipotensión a veces se presenta sin que haya choque, pero este por lo general se acom- paña de hipotensión.
  Cold, pale, moist, slimy skin due to sympathetic vasoconstriction, but may be warm in septic (distributive) shock.
  Pulse: rapid, filiform.
- Respiratory frequency: increased.

Jugular veins: engorged in cardiogenic shock and collapsed in hypovolemic shock.

Diuresis: oliguria as a manifestation of renal perfusion deficiency.

ETIOLOGY

A. Hypovolemic shock.

1. Loss of blood volume (acute hemorrhage).

External loss:

a) Trauma.

b) Gastrointestinal hemorrhage.

Internal loss (sequestered):

a) Hemothorax.

b) Hemoperitoneum.

c) Retroperitoneal hemorrhage.

d) Fractures.

2. Losses of plasma volume:

a) Serious burns.

b) Vomiting or diarrhea or both.

c) Diabetic ketoacidosis.

d) Diabetes insipidus.

e) Excessive use of diuretics.

f) Peritonitis.

g) Pancreatitis.

h) Splanchnic ischemia.

B. Cardiogenic shock.

1. Acute myocardial infarction.

2. Myocarditis.

3. Acute valve insufficiency.

4. Myocardial rupture.

5. Serious cardiac arrhythmias.

C. Distributive shock.

1. Septic shock.

2. Anaphylaxis.

3. Spinal cord trauma.

4. Anesthetics.

5. Narcotics and barbiturates.

D. Vascular obstructive shock.

1. Pulmonary embolism.

2. Pericardial tamponade.

3. Tension pneumothorax.

4. Obstruction of the vena cava.

5. Aneurysm dissection.

HEART FAILURE SYNDROME

CONCEPT

Heart failure is a functional disorder that is manifested by an inability of the heart to meet the metabolic needs of the body in some or all circumstances of normal life (or it does so by raising the filling pressure and that occurs in the course of the natural evolution of any heart disease with significant or prolonged hemodynamic repercussions.

This syndrome can be caused by a decrease in the contractile capacity of the heart or by an increase in pressure or volume overload imposed on the heart.
So it can be caused by systolic failure, diastolic failure, systodiastolic failure or by other factors that cause an impediment to ventricular filling. All of them will cause an increase in filling pressure with consequent venous congestion and deterioration of minute volume that support the clinical manifestations more in relation to water and visceral retention (the lungs, kidneys, liver) than with cardiac modifications as such. This gives it the connotation of a more or less generalized condition of the entire organism and can be considered a multisystem syndrome due to a primary anomaly of the heart that produces a series of hemodynamic and neurohormonal adaptations aimed at reestablishing normal cardiac function and eliminating symptoms in which dyspnea predominates with or without fluid retention.

NECESSARY CLASSIFICATIONS AND DEFINITIONS

Clinical classification according to predominance of affected ventricle

Clinics, experiments and hemodynamics agree in recognizing a generalized form of heart failure and partial forms that selectively affect the left or right heart. We will see this classification in more detail in the syndromography.

Classification according to the pathophysiological mechanism

Advances in the study of normal and pathological cardiac function, the development of Doppler echocardiography and the use of isotopes, as well as clinical-pathological observation itself have made it possible to establish systolic, diastolic and impeded ventricular filling heart failure, in addition to combined or systodiastolic. The practical importance of this pathophysiological distinction lies in the fact that its diagnosis, prognosis and treatment are different.

Very frequently (70-80% of cases) heart failure will be a consequence of the progressive deterioration of the nmyocardial contractile function, that is, a systolic failure that can range from the state of mild and moderate systolic dysfunction (subclinical) to that of marked systolic dysfunction that supports the clinical state of systolic heart failure.

In a smaller percentage (20-30%) a pressure overload is caused by the inability of the heart to distend or expand adequately and sufficiently during diastole to accept the ventricular volume, that is, there is diastolic failure. This disorder of distensibility, of the capacity to accept volume (compliance of the Anglo-Saxon literature) ranges from mild diastolic dysfunction to marked diastolic dysfunction that is expressed in the clinical state of diastolic heart failure. It can also be a combination of these two major previous pathophysiological types: systodiastolic.

In other circumstances, ventricular filling may be impeded due to causes unrelated to myocardial alterations, such as chronic pericardial constriction or pericardial effusion, mitral or tricuspid stenosis.

It is also important to clarify that on rare occasions acute heart failure can occur due to rupture of a leaflet or chordae tendineae without initial myocardial involvement.

Necessary definitions

Subclinical ventricular dysfunction. There is depression of ventricular function without symptoms.

Heart failure. It is a symptomatic ventricular dysfunction.

Myocardial failure, heart failure and circulatory failure. Congestive state. The terms myocardial insufficiency, heart failure and circulatory insufficiency are not synonymous, although in this same order each pathophysiological state is progressively more comprehensive and can include the other.

Myocardial failure has to reach a certain stage where it becomes more intense and, therefore, its dysfunction transforms into heart failure. However, there are situations in which a sudden hemodynamic overload of the heart or interference with filling without myocardial insufficiency causes heart failure.

For its part, heart failure always causes circulatory failure, however this can occur in non-cardiac disorders such as hypovolemic or anaphylactic shock, in the presence of a heart with normal or very slightly compromised function. The congestive state also called overload or congestion

Circulatory tion refers to excess blood volume that may be due to cardiac or non-cardiac causes.

The latter can be divided, in turn, into:

a) Disorders related to increased blood volume (excessive accumulation of salt and water due to steroids, excessive administration of blood and fluids, acute glomerulonephritis, renal failure).

b) Disorders related to an increase in venous return, a decrease in peripheral resistance or both (fistulas).

SYNDROMOGENESIS OR PHYSIOPATHOLOGY

The fundamental factors in determining cardiac output are heart rate and stroke volume. The latter, in turn, is determined by preload (ventricular volume at the end of diastole), contractility and afterload (given by the forces that oppose ventricular ejection).

In circumstances such as a myocardial anomaly with primary or secondary alteration of contractility, or a hemodynamic overdemand of volume or excessive pressure for ventricular function, a series of adaptation mechanisms are triggered that allow the cardiac pump to fulfill its function at least for some time.

Among the most important are:

1. Frank-Starling law in which reinforced preload helps maintain function since cardiac dilation during diastole increases the force and volume of systolic contraction.

2. Myocardial hypertrophy with or without dilation of the cardiac chambers and in which the contractile mass increases.

3. The activation of neurohormonal systems and especially the action of the sympathetic nervous system, reinforces contractility and increases heart rate, above all, with the release of noradrenaline (norepinephrine) in the cardiac adrenergic nerves. Likewise, the action of the renin-angiotensin-aldosterone system contributes to maintaining blood pressure and perfusion of vital organs.

Unfortunately, these mechanisms, which can be translated, among others, by tachycardia, dilation and hypertrophy, cause excessive compensation and become harmful; In this way, what they ultimately do is depress cardiac contractility and performance, as well as increase venous congestion.

In summary, the pathophysiological events and processes of heart failure are given by:

1. Modifications in myocellular genetic expression.

Starting point of a normal signal captured from the

heart disease, the genes that encode, among other factors, contractile proteins, mitochondrial structures and various angiotensin and dopaminergic receptors are modified; together with them, tissue growth factors are stimulated. All of this supports myocardial hypertrophy, the production of fibrosis and a lower dilation capacity of blood vessels. These phenomena are the basis of ventricular dysfunction. It is not yet known why some heart diseases induce structural changes that cause systolic or diastolic failure.

2. Hemodynamic alterations. Given by increased filling pressures, peripheral resistances and deterioration in minute volume as a consequence of ventricular dysfunction.

3. Neuroendocrine response. At the beginning there is an increase in plasma levels of the atrial natriuretic factor, which, as its name indicates, stimulates natriuresis and is also a vasodilator and inhibits the RAAS (renin-angiotensin-aldosterone system), but as myocardial deterioration progresses, the RAAS, which is antidiuretic and vasoconstrictor, predominates, natriuresis ceases and water and sodium retention occurs, with the corresponding symptoms. congestive.

4. Disorders in peripheral circulation and skeletal muscle that explain fatigue and poor exercise capacity.

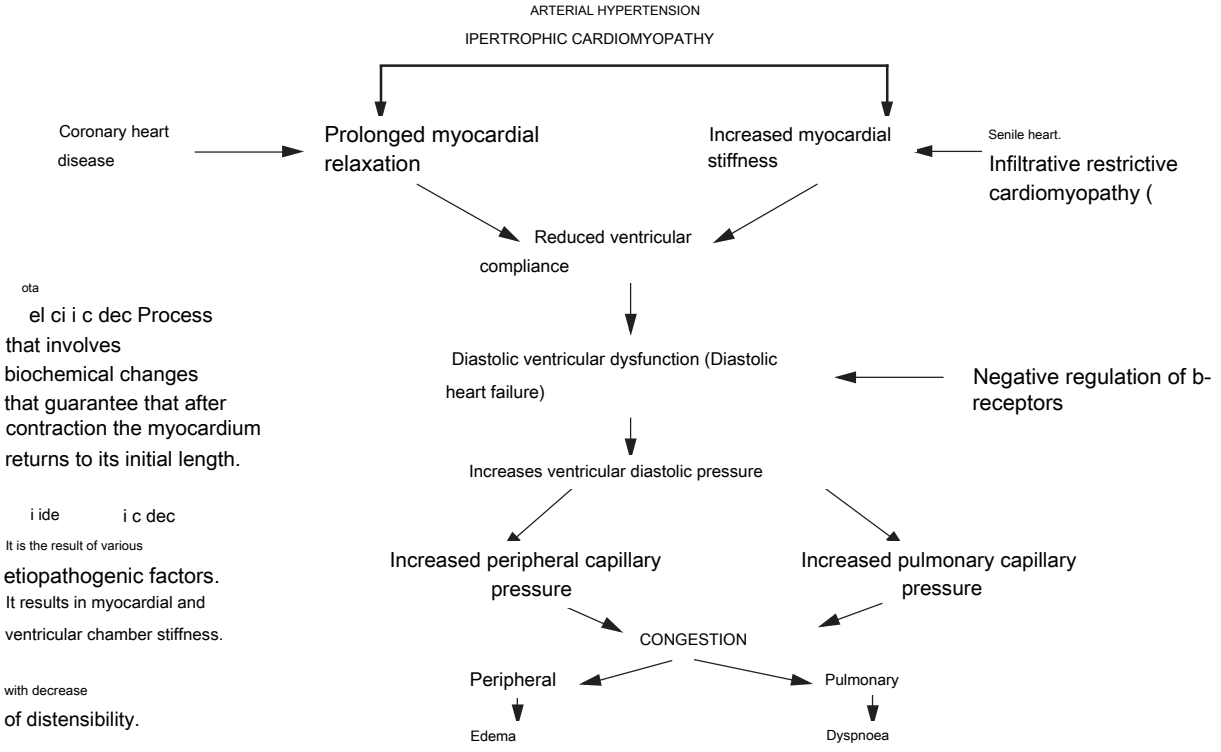
The defects of decreased cardiac contraction are:

1. Reduction in blood ejection during systole, with deterioration in minute volume; with decreased blood flow and tissue hypoperfusion (the so-called anterograde insufficiency).
2. The retrograde accumulation of blood returning to the heart, with blood stasis in different organs as a result of inadequate venous drainage due to a heart unable to push the blood it receives towards the pulmonary artery (so-called retrograde insufficiency), causes congestion in the pulmonary territory (left heart failure) and in the systemic territory (right heart failure).

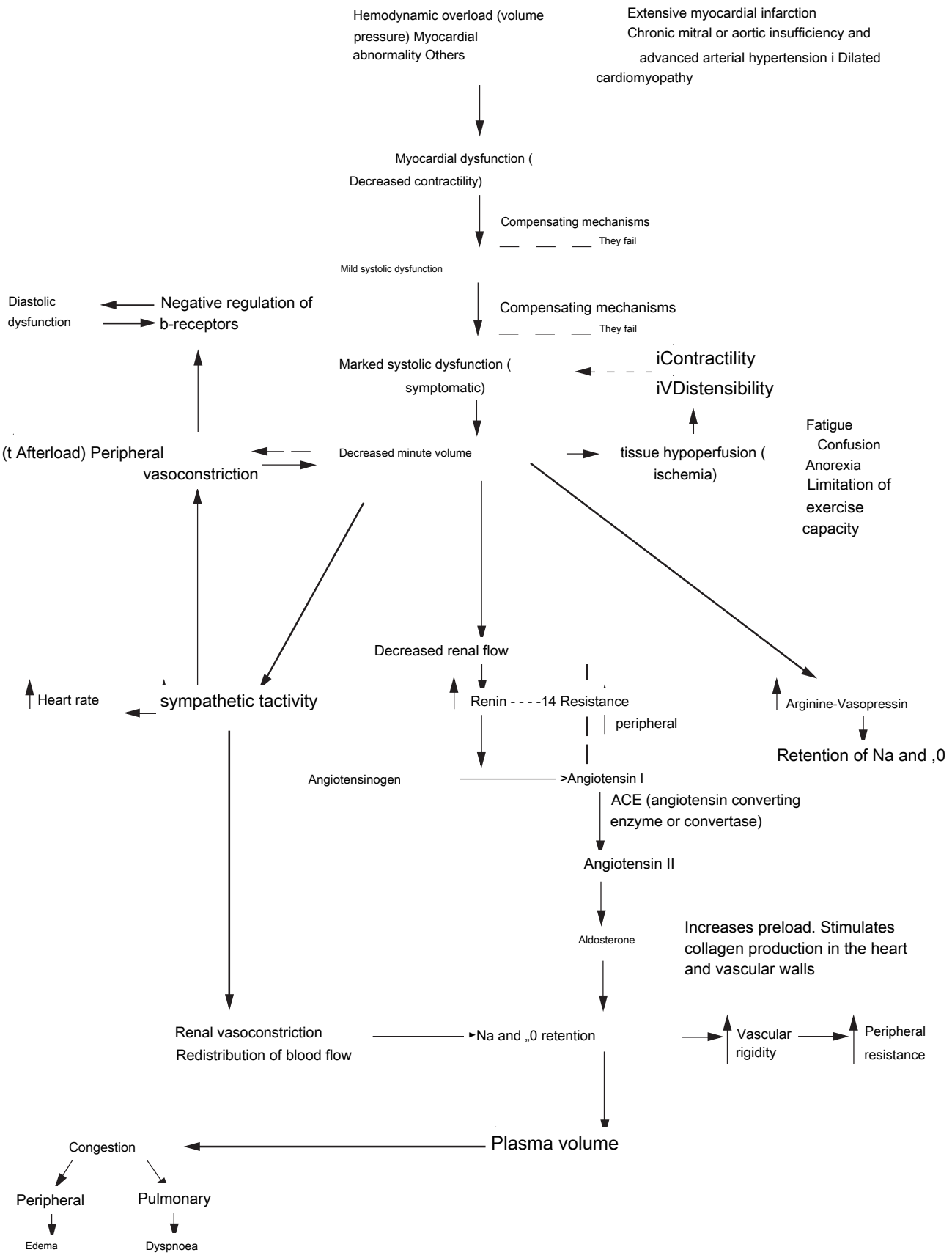
From these effects of the deterioration of the minute volume with hypoperfusion and venous congestion intertwined with the compensation mechanisms, becoming harmful, the symptoms and signs of heart failure arise.

Below we summarize in a diagram the main pathophysiological processes involved in heart failure, especially systolic, as well as the most significant diastolic, without forgetting their possible association.

Pathophysiology of heart failure with diastolic ventricular dysfunction


ARTERIAL HYPERTENSION
IPERTROPHIC CARDIOMYOPATHY
Coronary heart disease
Prolonged myocardial relaxation
Increased myocardial stiffness
Senile heart.
Infiltrative restrictive cardiomyopathy (
Reduced ventricular compliance
ota
el ci i c dec Process that involves biochemical changes that guarantee that after contraction the myocardium returns to its initial length.
Diastolic ventricular dysfunction (Diastolic heart failure)
Negative regulation of b-receptors
i ide i c dec
It is the result of various etiopathogenic factors.
It results in myocardial and ventricular chamber stiffness.
Increases ventricular diastolic pressure
with decrease of distensibility.
Increased peripheral capillary pressure
Increased pulmonary capillary pressure
CONGESTION
Peripheral
Pulmonary
Edema
Dyspnoea

Pathophysiology of heart failure with systolic ventricular dysfunction



CLINICAL CLASSIFICATION

Three types of heart failure can be distinguished clinically:

1. Left ventricular failure, with predominantly respiratory manifestations.
2. Right ventricular failure, characterized by the predominance of liver and kidney signs.
3. Global heart failure, in which pulmonary, hepatic and renal manifestations come together.

The syndromographic description that follows generally corresponds to systolic heart failure.

Left ventricular failure

When the left ventricle fails for any reason, a stasis occurs in the blood from the lung, which gives rise to predominantly pulmonary symptoms.

Syndromography or positive diagnosis (fig 45 1)

Clinical picture

1. Tachycardia. It is one of the first signs of cardiac decompensation; The insufficient heart increases its frequency as a compensatory mechanism.
2. Dyspnea on exertion. A certain degree of dyspnea on exertion is normal in people with a lack of training.

Fig. 45.1 Left ventricular failure.

physical in obese; However, a marked increase in dyspnea on exertion is significant for pulmonary congestion.

3. Orthopnea, cardiac pseudoasthma and acute lung edema. These states depend on increasing degrees of pulmonary congestion and are therefore interrelated.

Vital capacity is reduced when the patient is lying down; When this reduction is small, the orthopneic position manages to overcome the sensation of suffocation. Paroxysmal cardiac dyspnea, with or without wheezing (cardiac asthma), usually occurs at night, and the patient often achieves relief by sitting for half an hour or more. Sitting increases pressure in the right atrium, which can overload the right ventricle, thereby decreasing left ventricular tension.

Acute lung edema presents the same signs as cardiac asthma, but in extreme degrees; The patient begins to cough and produces frothy pink expectoration; The orthopnea position fails to alleviate the shortness of breath. He usually suffers great panic as the feeling of imminent suffocation becomes more vivid. Wet rales are heard throughout the chest. The state of panic makes the administration of morphine urgent and is the most useful measure to alleviate these disorders.

4. Bilateral basal rales. The finding of wet rales at the lung bases is one of the most important signs in left ventricular failure. They are the typical opponents of pulmonary congestion and can be crackles or subcrackles. Pulmonary stasis is often accompanied by stasis in the pleural veins, which can cause hydrothorax.

5. Displaced tip beat. The apex beat moves downward and to the left indicating an increase in the size of the left ventricle.
6. Gallop rhyme. When the left ventricle fails, there is often an unmistakable galloping sound (this is a three-beat rhythm with a galloping cadence) that is best heard at the tip or slightly to the right of the tip.

7. Alternating pulse. It is also an unmistakable sign of left ventricular failure.
8. Cheyne-Stokes breathing. This breathing, characterized by alternating periods of hyperpnea and apnea, results from cerebral ischemia and often follows left ventricular failure. It has already been studied in detail.

Complementary exams

Radiographic examination. The radiographs show engorgement of the pulmonary vessels at the level of the hilum and hypertrophy of the left cavities.

Electrocardiogram. Its alterations depend on the etiology; Mitral P waves may be observed, other times signs of left ventricular hypertrophy.

Type M and two-dimensional echocardiography including Doppler study. This study is definitive in the diagnosis of heart failure because it allows the measurement of the left ventricular ejection fraction, which when less than 45% can be accepted as proof of left ventricular dysfunction.

Cardiac catheterization. It remains useful for evaluating valvular heart disease and measuring pressures.

Scintigraphy. It is very useful to evaluate myocardial perfusion and contractility, as well as areas of ischemia and fibrosis.

Stress test. It may be important to evaluate functional impairment and the severity of symptoms.

Other techniques. Computed axial tomography, nuclear magnetic resonance, and positron emission tomography are very useful, but very expensive, tests.

cough

tiology

Left ventricular failure is usually due to: high blood pressure, aortic valve disease (stenosis or insufficiency), mitral valve disease (insufficiency), and coronary artery disease. In all cases there is a disorder in myocardial nutrition as a result of myocardial ischemia.

Right ventricular failure fig

It is rarely isolated and pure; In general, it is almost always integrated into global heart failure, which may be established from the outset, or may follow a period of left ventricular failure of variable duration.

In these cases, the blood pool is established in the two vena cavae and, consequently, venous hypertension and edema will predominate in the symptoms.

Syndromography or positive diagnosis

Clinical picture

1. Painful hepatomegaly. It is the earliest objective symptom of right ventricular failure. It generally exceeds the costal margin by several centimeters. The liver is firm and painful.
   When it is continuously compressed, it can cause venous engorgement in the neck (hepatojugular reflux).

   It precedes peripheral edema well in advance.

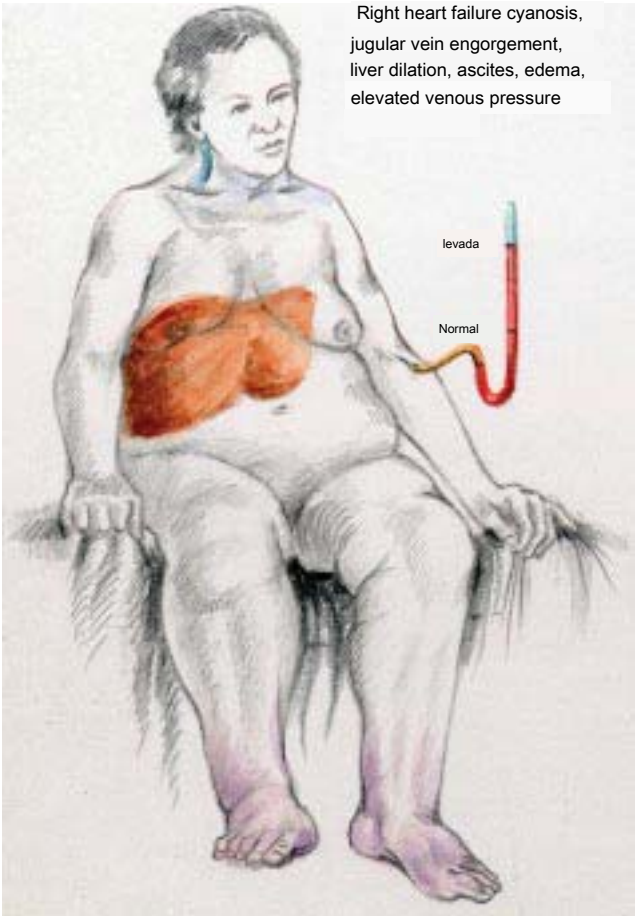

Right heart failure cyanosis, jugular vein engorgement, liver dilation, ascites, edema, elevated venous pressure
levada
Normal

Fig. 45.2 Right ventricular failure.

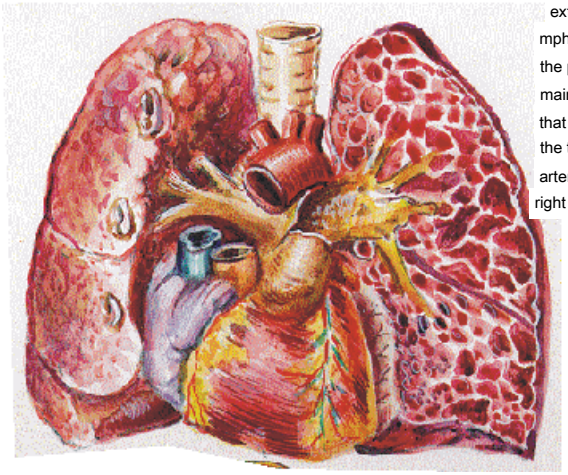
2. Oliguria. It is also early, it can decrease to 400 mL or less per day. It is very concentrated and the density reaches or exceeds 1,025.
3. Peripheral edema. They manifest in the most advanced stages of right ventricular failure and are always present when venous pressure exceeds 25 cm of water. They are preceded by a state of preedema with an increase in body weight of 1-5 kg.
   The characteristics of this type of edema have already been studied.
4. Engorgement of the neck veins. It increases in the lying position and with liver compression.

Complementary exams

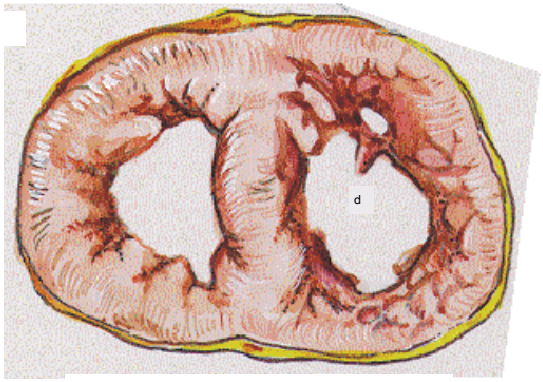
Urine. Demonstrates albuminuria. There may be urea retention between 60 and 100 mg/100 mL.

Radiographic examination. It may show hypertrophy and/or dilation of the right ventricle and atrium, especially in an oblique position. Sometimes there are chronic lung lesions if that has been the cause.

Electrocardiogram. Right atrial and ventricular hypertrophy is particularly clear in cor pulmonale and congenital heart disease.



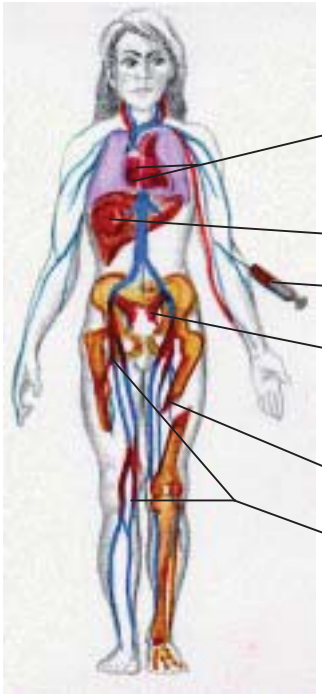
extensive pulmonary
mphisema with great distention of
the pulmonary trunk and
main pulmonary arteries
that compress the aorta against
the trachea pulmonary
arteriosclerosis and
right ventricular



Hypertrophy and dilation of the right ventricle

Fig. 45.3 Chronic cor pulmonale.

Fig. 45.4 Acute cor pulmonale.



-Thrombus in the right
heart (septal infarction,
atrial fibrillation)

tumor emboli air

embolism

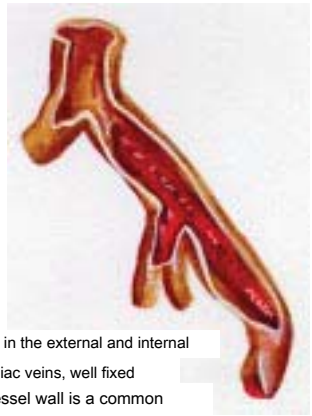
-Thrombus in the
pelvic veins (
after pelvic or
prostate

Fat emboli (
fractures)

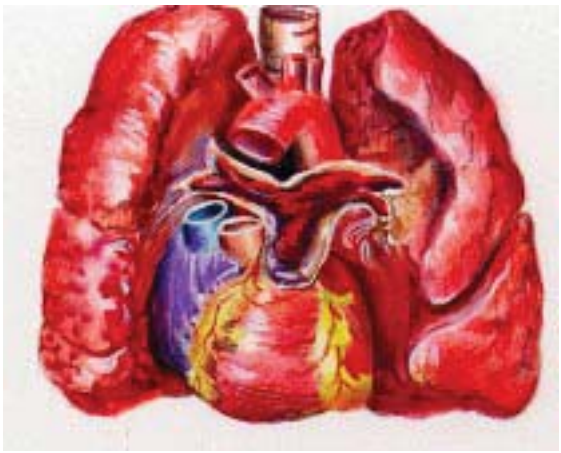
Thrombus in the
deep veins of
the lower extremities

Sources of
pulmonary

multiple plungers
in the lung tree
arterial with acute
infarctions



Thrombus in the external and internal
common iliac veins, well fixed
to the vessel wall is a common
source of pulmonary embolism



massive embolism of the pulmonary trunk
and the main pulmonary arteries
no heart attack, dilated right heart

Echocardiogram. What has already been stated in left ventricular failure is generally valid. It allows recognition of right atrial and ventricular hypertrophies, as well as some congenital heart diseases.

tiology

Right ventricular failure frequently follows left ventricular failure. The

Chronic lung diseases such as emphysema and primitive pulmonary arterial hypertension cause the condition known as chronic cor pulmonale (Fig. 45.3). When pulmonary artery hypertension sets in suddenly (pulmonary thromboembolism, for example), the right ventricle fails in the same way, causing acute cor pulmonale (Fig. 45.4).

clinical, imaging, electrocardiographic, etiological, differences between heart failure with systolic diastolic dysfunction

	Systolic heart failure	Diastolic heart failure
Frequency	From 70-80	From 20-30
re e taci cl ic	Chronic, progressive dyspnea	Sudden, paroxysmal
	Slow-onset pulmonary edema	dyspnea Acute pulmonary edema with small
	Frequent malleolar edema	Less common malleolar edema
A tecede te		
coronary heart disease		
arterial hypertension		
Diabetes mellitus		
Valvulopathy		
Paroxysmal dyspnea		
xame ico		
Cardiomegaly		Dilated ventricle Not
Tip Beat	Displaced	displaced
Muffled heart sounds		
Gallop R3 (3rd noise)		
Gallop R4 (4th noise)		
arterial hypertension		
Mitral regurgitation		
Rattles		
Edema		
Jugular vein distension		
X me e compleme tario		
di f de		
Cardiomegaly		
Pulmonary congestion		
lec C di		
Low voltage VI		
Waves		
CC di		
reduced LVEF	(45)	(45)
LV dilation		
VI		

ont

	Systolic heart failure	Diastolic heart failure
thiology m recue te	Extensive infarction Dilated cardiomyopathy Chronic valve regurgitation evolved arterial hypertension	concentric hypertrophy (high blood pressure, hypertrophic cardiomyopathy) Ischemic heart disease (without acute or small myocardial infarction) - Restrictive cardiomyopathy

VI: left ventricular hypertrophy.
LVEF: Left ventricular ejection fraction.
VI: Left ventricle.

CORONARY INSUFFICIENCY SYNDROME

CONCEPT

Coronary insufficiency (ischemic heart disease) is the name given to the clinical and electrocardiographic manifestations that result from the acute or chronic, temporary or permanent decrease in blood flow to the heart.

SYNDROMOGENESIS PATHOPHYSIOLOGY

It is of primary importance to remember that coronary irrigation alone is insufficient in relation to the needs of the myocardium to carry out its work. Coronary irrigation may be sufficient to satisfy the needs of the myocardium at rest, but unable to cover them when the work of the heart increases due to exercise, emotions, fever, etc. In such circumstances, myocardial ischemia occurs and clinical manifestations appear: precordial or retrosternal discomfort or pain. On the other hand, pain may appear in conditions of rest, if there is a sudden decrease in blood flow to a branch of the coronary arteries.

Coronary insufficiency syndrome is due in most cases (more than 90%) to atheromatous occlusion of the coronary arteries, and its prevalence in our adult population is relatively high.

Other mechanisms that can cause this syndrome

Are:

1. Volume overloads. Aortic insufficiency, mitral insufficiency.

2. Pressure overloads. High blood pressure, aortic stenosis.

In these two groups, myocardial oxygen demands increase considerably.

3. Reduction of oxygen supply. Anemias, high altitudes, chronic cor pulmonale.
4. Conditions that occur with low minute volume. Mitral stenosis, pulmonary stenosis, cardiomyopathies.
5. Rhythm disorders. Paroxysmal tachycardia.

We believe it is necessary to insist that, in the vast majority, coronary insufficiencies are caused by atheromatosis of the nutrient arteries of the myocardium.

CLASSIFICATION

We will consider here some types of insufficiency coronary:

- Angina pectoris.
- Myocardial infarction.
- Coronary insufficiency without angina.
-Other forms of atherosclerotic heart disease, such as: atrial fibrillation, significant ventricular extrasystole, complete block of the left bundle branch and left anterior hemiblock.

Of these four groups we will limit ourselves at this moment to explaining only the first two, since the others will be studied in Internal Medicine.

angina pectoris

Concept

Syndrome characterized by paroxysmal pain of retrosternal location, with characteristic irradiation, which is triggered by exercises, emotions or other states in which cardiac work increases, and which is relieved by rest and nitroglycerin.

Syndromography or positive diagnosis

Clinical picture

1. Pain located retrosternally or precordially, oppressive or compressive, that appears during or immediately after an effort, radiates mainly to the left arm, is relieved with rest and disappears almost always in less than 10 minutes or with the administration of nitroglycerin.
   If the pain does not present with the stereotypical characteristics indicated, and appears at rest or after mild exertion, with prolonged duration, or if it is the first time it occurs, it is called "unstable angina."

2. Feeling of anguish or imminent death.

3. Gastrointestinal symptoms: nausea, vomiting, etc.

ra.

4. Other symptoms: dyspnea, palpitations, paleness, sweating. Sometimes physical signs of the underlying disease appear.

Complementary exams

The electrocardiogram may show, during pain, depression of the ST segment of 1 mm or more with a flattened or negative T wave.

These electrocardiographic alterations may return to normal after the pain disappears, or they may persist as an expression of sustained myocardial ischemia.

There is no leukocytosis, the erythrocyte sedimentation rate is normal and the glutamicoxaloacetic transaminase, as well as other enzymes, are normal.

tiology

Determining factors. Atherosclerotic narrowing of the coronary arteries in more than 90% of cases.

Predisposing factors. There are numerous risk factors that predispose to suffering from this syndrome. Among them it is necessary to highlight: high blood pressure, smoking, obesity, hyperlipidemia, diabetes mellitus, diet rich in saturated fats, psychosocial tension (emotional stress) and sedentary life.

Triggering factors. Physical efforts, emotions, cold, intercourse, etc.

Myocardial infarction

Concept

Myocardial infarction is the clinical, electrocardiographic and humoral syndrome produced by ischemic necrobiosis of a sector of the myocardium, and which is characterized by sustained retrosternal pain or oppression, arterial hypotension or shock, together with characteristic progressive electrocardiographic alterations, fever and increase in certain enzymes in the blood.

Syndromography or positive diagnosis

Clinical picture

1. Retrosternal or precordial pain. Sometimes it is located in the epigastrium and is constrictive, distressing, usually very intense and radiating to the left shoulder and arm. It usually starts suddenly and lasts more than 30 minutes. It does not undergo alterations nor is it modified by changes in position, breathing, nitroglycerin or rest.

2. Central circulatory symptoms. Decrease in the intensity of both heart sounds, tachycardia and sometimes gallop rhythm. Arterial hypotension that can lead to shock. Presence of extrasystoles and other arrhythmias. Pericardial rub is present in 10% of cases.

3. Peripheral circulatory symptoms. Rapid and weak pulse, sweating, cold skin and paleness: these symptoms are more evident when the infarction is complicated by shock (cardiogenic).

4. Respiratory symptoms. Dyspnea and sometimes wet rales at the lung bases when the infarction is accompanied by heart failure.

5. Digestive symptoms. Sometimes nausea and vomiting.

6. General symptoms. A fever may occur.

Complementary exams

Hemogram. Discrete leukocytosis, sometimes moderate, after the first 24 hours.

Erythrosedimentation. Accelerated in the first 3-4 days.

Glutamicoxalolacetic transaminase. It rises during the first 48 hours. Lactic dehydrogenase and phosphocreatine kinase may also be elevated (see p. 562).

Electrocardiogram. Appearance of a deep Q wave (more than 30% of R) and wide (0.04 s or more) with positive displacement (more than 1 mm) of the ST segment and inverted and symmetrical wave in certain leads depending on the location of the lesion.

tiology

The causes that produce the infarction are the same as those we have pointed out in angina pectoris when the lesion is more intense and causes complete obstruction of the coonary arteries or their branches.

ARTERIAL IPERTENSION SYNDROME

CONCEPT

High blood pressure (HTN) can be defined as an increase in arterial blood pressure (BP) above normal values, taking the World Health Organization (WHO) criteria as maximum limits (see Chapter 12, p. 143).

SYNDROMOGENESIS OR PHYSIOPATHOLOGY

We will briefly summarize the factors that have been invoked to produce an increase in blood pressure in the intimate mechanism of arterial hypertension, both symptomatic and essential:

- Circulatory factors.

- Water and salt retention.

- Kidney factors.

– Decreased response of the baroreceptors.

Renin-angiotensin system.

Circulatory factors

1. Cardiac output.

2. Total peripheral resistance.

If cardiac output increases, blood pressure will rise unless there is a corresponding decrease in peripheral resistance. The increase in the latter takes place mainly in the arterioles and is due to the contraction of their muscular walls.

Between these two factors, the most important is the elevation of peripheral resistance, which is the one most uniformly observed in the presence of arterial hypertension.

We will find the increase in cardiac output as an important factor in arterial hypertension in renal failure, toxemia of pregnancy and acute glomerulonephritis. For the most part, patients with these processes are very sensitive to fluid overload, which is why sudden elevations in blood pressure may occur.

The causes of elevated peripheral resistance are unknown in most hypertensive patients, except in cases of pheochromocytoma, in which high levels of catecholamines are demonstrated in the plasma, or in cases of hemangiopericytoma of the kidney in which high levels of renin and angiotensin can be demonstrated.

It is believed that high blood pressure itself causes changes in peripheral resistance, increasing the contraction of the vessels.

Some authors consider that the sequence of events is as follows: retention of salt and water due to a kidney disorder that leads to an increase in cardiac output, which would lead to an increase in blood pressure and consequently contraction of the peripheral arterioles with an even greater increase in blood pressure; and in this way the cycle repeats.

Other authors consider that there is a primary cause of increased peripheral resistance, which would probably cause arterial hypertension.

Water and salt retention

It has already been mentioned that water overload is poorly tolerated by patients with kidney failure, in whom it causes an increase in blood pressure;
This overload improves with the removal of body fluid and restriction of salt in the diet, or with nephritis.

tomy that is performed in many patients before performing a kidney transplant.

Experimentally, "renopriva hypertension" has been tested; More precise studies seem to indicate that hypertension that develops in the absence of kidneys most likely depends on an increased sensitivity to water and salt overload. Experimental work and also observation in humans seem to demonstrate the possible existence of a genetic factor in relation to this sensitivity to salt. This relationship between water and salt metabolism and hypertension is very closely linked to the excessive excretion of adrenal corticosteroids.

Kidney factors

That the kidney is a direct and primary cause of some cases of hypertension is demonstrated by its cure by unilateral nephrectomy, when one of the kidneys is injured. The cause of the mechanism that produces hypertension in these cases seems to be interference with the kidney's blood supply.

Below we will present some of the main renal factors that have been pointed out in the production of hypertension.

1. Inability to eliminate or destroy an extrarenal pressor substance.
2. Inability to produce a vasodilatory substance, mainly prostaglandin such as type E (PGE).

3. Increased response to a normal pressor mechanism, mainly through the sympathetic nervous system. It has been shown that the substance that has the greatest stimulating action is angiotensin.
The degree of activity of the sympathetic nervous system can be determined by the finding in the circulation of norepinephrine and dopamine beta-hydroxylase, substances released by the activity of this nervous system. Both substances are increased in high blood pressure.

Decreased response of baroreceptors

Injury to the baroreceptors located in the aorta and carotid sinus alters their response and, as a consequence, blood pressure rises.

Renin angiotensin system

Renin is stored in the form of granules in the smooth muscle fibers of the walls of the afferent arterioles of the glomerulus, adjacent to the macula dense.

It is probably released bound to a protein that reacts with the substrate to first produce the decapeptide angiotensin I, which is mainly converted.

ly in an octapeptide pressor: angiotensin II (by hydrolysis of two amino acids) when passing through the lung or other tissues.

Angiotensin II is the most powerful vasoconstrictor and acts directly on smooth muscle, resulting in a reduction in renal blood flow.

We must also point out the existence of renin-producing tumors (such as hemangiopericytoma) that cause arterial hypertension and give rise to high levels of aldosterone with a decrease in potassium in the plasma.

It is worth noting the importance of correlating various factors, adding to those already mentioned, the nervous system, hormonal and psychic influences and the role of heredity.

Arterial hypertension of unknown cause has been considered as an expression of reaction between the internal environment and the external environment, thinking that the increasing increase in external influences would exceed in intensity and frequency the capacity of the internal environment to maintain tension stability.

Figure 45.5 shows schematically the different factors that intervene in the production of arterial hypertension.

CLASSIFICATION

There is a syndrome of high blood pressure in which the doctor cannot find a cause capable of determining it;
This hypertension, which is the most common, is called primitive, essential or idiopathic arterial hypertension.

In addition to this type of hypertension, there is another group in which we find a disease capable of causing it: it is symptomatic or secondary hypertension.

Sometimes it is nephropathy or any of the causes that we will list later. The advancement of modern diagnostic procedures has decreased the percentage of "essential hypertension"

OTHER CLASSIFICATIONS

From an evolutionary point of view, arterial hypertension is classified according to WHO criteria into the following stages or stages:

Stage I. Elevated pressure according to established criteria, without evidence of organic alterations in the cardiovascular system.

Stage II. Elevated pressure with hypertrophy of the heart, minimal vascular alterations in the fundus, or both at the same time, but without evidence of lesions in other organs.

Stage III. High pressure with injuries to various organs (heart, eye, kidney, brain or others).

From the point of view of the nature of hypertension, have:

1. Permanent hypertension. There are high figures, with evident continuity.
2. Paroxysmal hypertension. It is found in pheochromocytoma, toxemia of pregnancy and colic of lead poisoning.

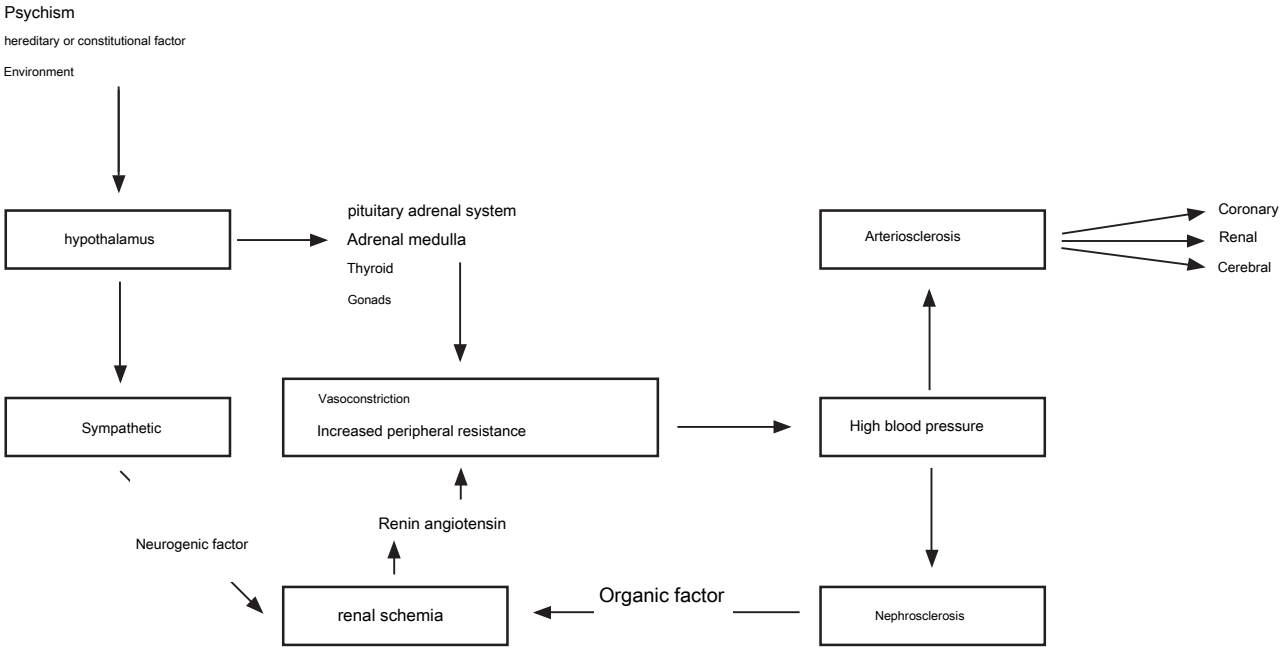


Fig. 45.5 Scheme of the different factors involved in the production of arterial hypertension.

From the point of view of patient inspection,

have:

1. Red or plethoric hypertensives. They correspond to the short-line constitutional type and the obese one.
2. Pale hypertensives. In them, hypertension is sometimes associated with the normal constitutional type. It is observed in those affected by arteriosclerosis and kidney patients.

From the point of view of which tension figure is altered, we have:

1. Systolic hypertension. The maximum number is altered and the minimum is normal (good prognosis).
2. Diastolic hypertension. The minimum is elevated and the maximum is normal (worse prognosis).
3. Systodiastolic hypertension. Both pressures are elevated; There are usually cerebrovascular, renal and cardiovascular complications (poor prognosis).

Criteria for high blood pressure in older adults to you or

C	i e e i (	e	lic
lic Stage 1	Discrete) (	140-159	90-99
Stage 2	Moderate) (	180-179 OR 100-109 Stage 3	
180-209 or	Severe)	110-119 Stage 4 (Very severe)	
210 and	more or 120 and more Suitability for Cuba (1998).		

Based on the average of two or more readings taken on each of two or more visits after the initial scrutiny. When the systolic and diastolic pressure figures fall into different categories, the highest is taken to assign the classification category.

SYNDROMOGRAPHY 0 POSITIVE DIAGNOSIS

The criteria for arterial hypertension have been indicated previously. Any person who, in three occasional feedings in a sitting position, presents figures above the stated criteria, should be considered hypertensive.

Some authors also add standing after 3 minutes in that position.

When only the maximum is altered, we speak of systolic hypertension; if the minimum is altered, diastolic hypertension. When both tensions rise, we speak of systodiastolic hypertension.

The increase in maximum pressure is important, but the increase in minimum pressure is of much more value, since serious and serious accidents of arterial hypertension are almost always linked to the increase in diastolic pressure.

Clinical picture

Each hypertensive patient has his or her own symptoms. We can assure, and the example has never been clearer, that there are no diseases, only sick people. Many patients have high blood pressure and remain totally ignor before it, until a day when, when a clinical examination is performed, the high blood pressure level can be found. Other times it debuts due to a serious complication (heart attack, stroke, etc.).

The study of the clinical picture of arterial hypertension corresponds to Internal Medicine, but we can say that this picture is extraordinarily polymorphous in its manifestations and that it will depend on the severity of the disease, the cause and the presence of cardiac, neurological and renal complications, fundamentally.

During the physical examination we will verify, in addition to the blood pressure levels, the signs of the impact of this hypertension on the cardiovascular system (left ventricular hypertrophy) and other signs such as disorders in the elimination of urine, retention of urea or creatinine, alterations of the arterioles in the fundus of the eye with or without papilledema, etc.

Complementary exams

The most commonly used examinations for study are:

- Simple or contrasted x-rays (urograms, arteriography, etc.) .
- Electrocardiogram, ultrasound and kidney function tests, fundus examination, dosage of hormones or their excretion products and special tests according to the cause of high blood pressure.

ETIOLOGY

We must differentiate essential arterial hypertension from those secondary to well-known or defined diseases or causes (Fig. 45.6).

According to its etiology, arterial hypertension is distributed as follows (Guédon):

Essential TAs	60
Secondary TAs Renal:	40
Parenchymal nephropathies.	25
Renal vascular lesions	12
Pheochromocytoma	0.5
Conn syndrome	1
Coarctation of the aorta Various	0.5 1

Renal causes are the most common among secondary arterial hypertension. Theoretically, renal arterial hypertension can be caused by kidney diseases; Among these, the main ones are: glomerulonephritis in all its stages of evolution, diabetic glomerulopathy, interstitial nephritis (gout, hyperparathyroidism, chronic pyelonephritis, polycystosis, etc.) and finally, anomalies of the renal arteries, which are frequent and responsible for arterial hypertension called renovascular.

CA SAS D HP RT NS NS C NDAR A P DNSR TRATABLE S R RG CAM NT

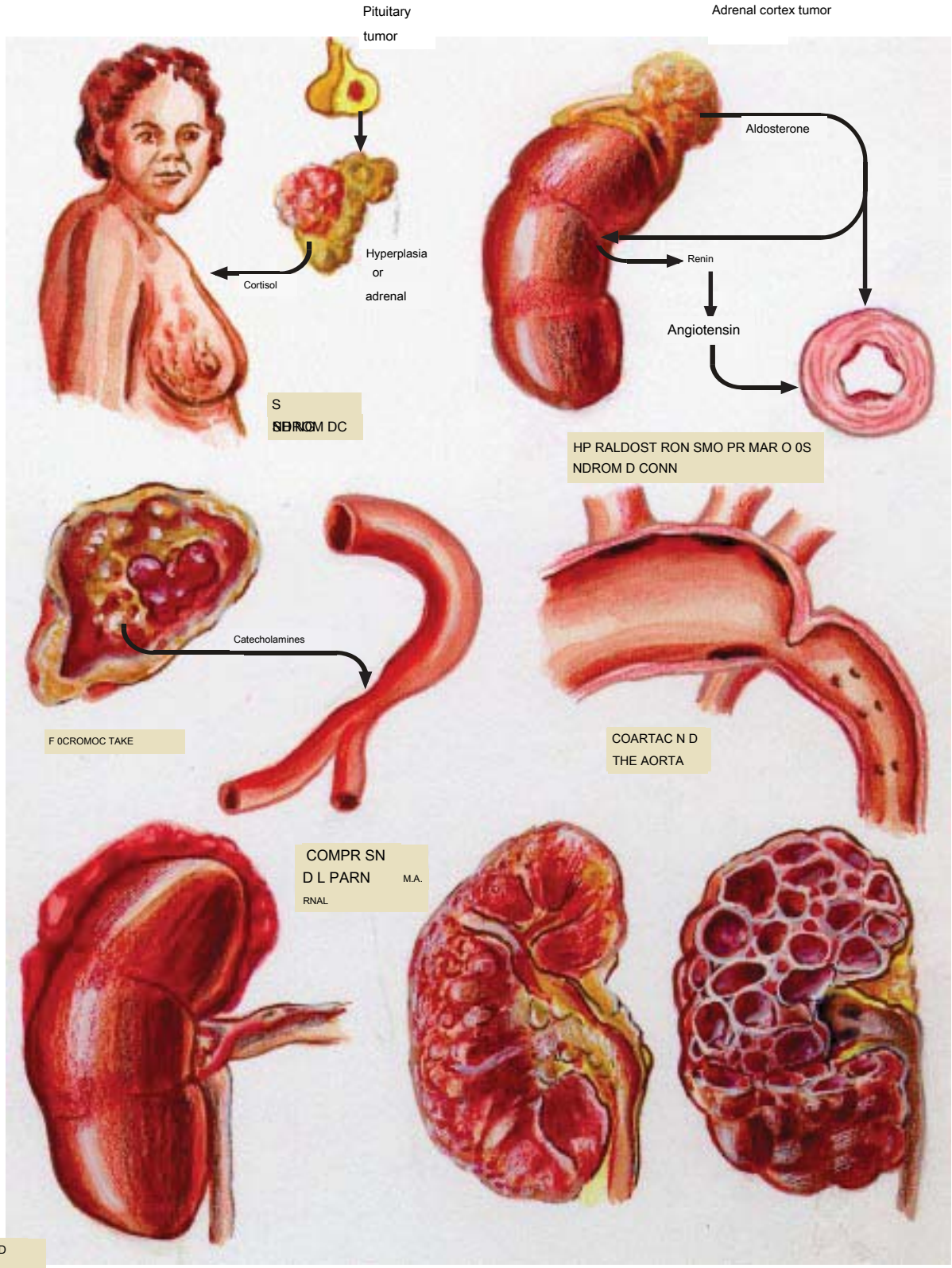

Pituitary tumor
Adrenal cortex tumor
Hyperplasia or adrenal
Cortisol
Aldosterone
Renin
Angiotensin
Catecholamines
F 0CROMOC TAKE
COARTAC N D THE AORTA
COMPR SN D L PARN M.A. RNAL
NF RM DAD 0 BUY Y N D THE ART RA RNAL
P LON FR TS NLAT RAL O R N POL ST CO

Fig. 45.6 Secondary arterial hypertension.

We would like to make it clear that until around 1985, in the best centers, essential arterial hypertension represented more than 90%, but the development of science has made it possible to reduce this percentage; However, this reduction is only obtained in those centers where all the modern resources are available, so we can consider that in general practice, essential arterial hypertension still occupies a figure close to 90% of all cases, especially when we consider that 15 to 30% of the adult population has blood pressure levels above normal limits and, however, the statistics published in books refer fundamentally to the patients who go to hospitals or polyclinics for already presenting symptoms in relation to their hypertensive condition. This means that in large epidemiological surveys, where a large symptomatic or asymptomatic population sample is studied, the results show that the percentage of essential blood pressure is 90 or more.

VALVULAR SYNDROMES

From a clinical point of view we can distinguish the following valvular syndromes:

Mitral stenosis.

- Mitral insufficiency.
- Mitral valve prolapse.
- Tricuspid stenosis.
- Tricuspid insufficiency.
- Aortic stenosis.
- Aortic insufficiency.
- Pulmonary stenosis.
- Lung failure.

MITRAL STENOSIS FIG.

Concept

It consists of the narrowness of the mitral valve orifice, which makes it difficult for blood to pass from the left atrium to the left ventricle during diastole.

Syndromog nesis or pathophysiology

The mitral leaflets are rigid, thick and fused to a variable degree with the chordae tendineae, also rigid and shortened; They make it difficult for blood to pass from the left atrium to the left ventricle.
Blood accumulates in the left atrium and, through a retrograde route, causes hypertension in the pulmonary circulation, which lacks valves, causing pulmonary hypertension. All of this will have an impact on the right ventricle and it, in turn, will end up dilating, while that the left ventricle reduces in size by receiving less blood than usual. The enormous size of the left atrium compresses the esophagus (which can cause dysphagia), the trachea and bronchus (cough), and more rarely the left recurrent nerve (bitonal voice).

Syndromography or positive diagnosis

Clinical picture

1. Dyspnea, which depends on the degree of stenosis.
2. Frequent hemoptysis and palpitations that depend on hypertension in the pulmonary bed and artery.
3. Facies and hands with cyanosis.
4. Murmur with a low tone and harsh timbre. The Duroziez rhythm includes: the drag or rumble (rru), the presystolic murmur (fu), the brilliance of the first noise (tt) and after a short silence the doubling of the second noise (ta-ta): rrufutt-tata.

Complementary exams

Radiology. In the right anterior oblique position (RAO) there is a projection of the middle arch forward and the left atrium backward. Kerley lines can be observed due to edema and lymphatic engorgement of the pulmonary interlobular septa.

Cardiac catheterization. Low minute volume. In the left atrium there is an increase in mean pressure (equal to or more than 20 mm Hg). There is hypertension in the pulmonary capillary (greater than 15 mm Hg), as well as in the pulmonary artery and right ventricle.

Electrocardiogram. Mitral P waves, right ventricular hypertrophy and overload, and frequent atrial fibrillation.

count.

Etiology

Pure rheumatic mitral stenosis (exceptionally congenital), stenosis plus rheumatic regurgitation (mitral disease).

MITRAL INSUFFICIENCY FIG.

Concept

It is the inability of the valve to keep the left atrioventricular orifice closed during systole, which results in blood reflux toward the atrium and the subsequent hypertrophy and dilation of that cavity.

Syndromog nesis or pathophysiology

Because of mitral incompetence, blood regurgitates from the left ventricle into the left atrium, because during systole the pressure is lower in the atrium than in the aorta. Heart failure,

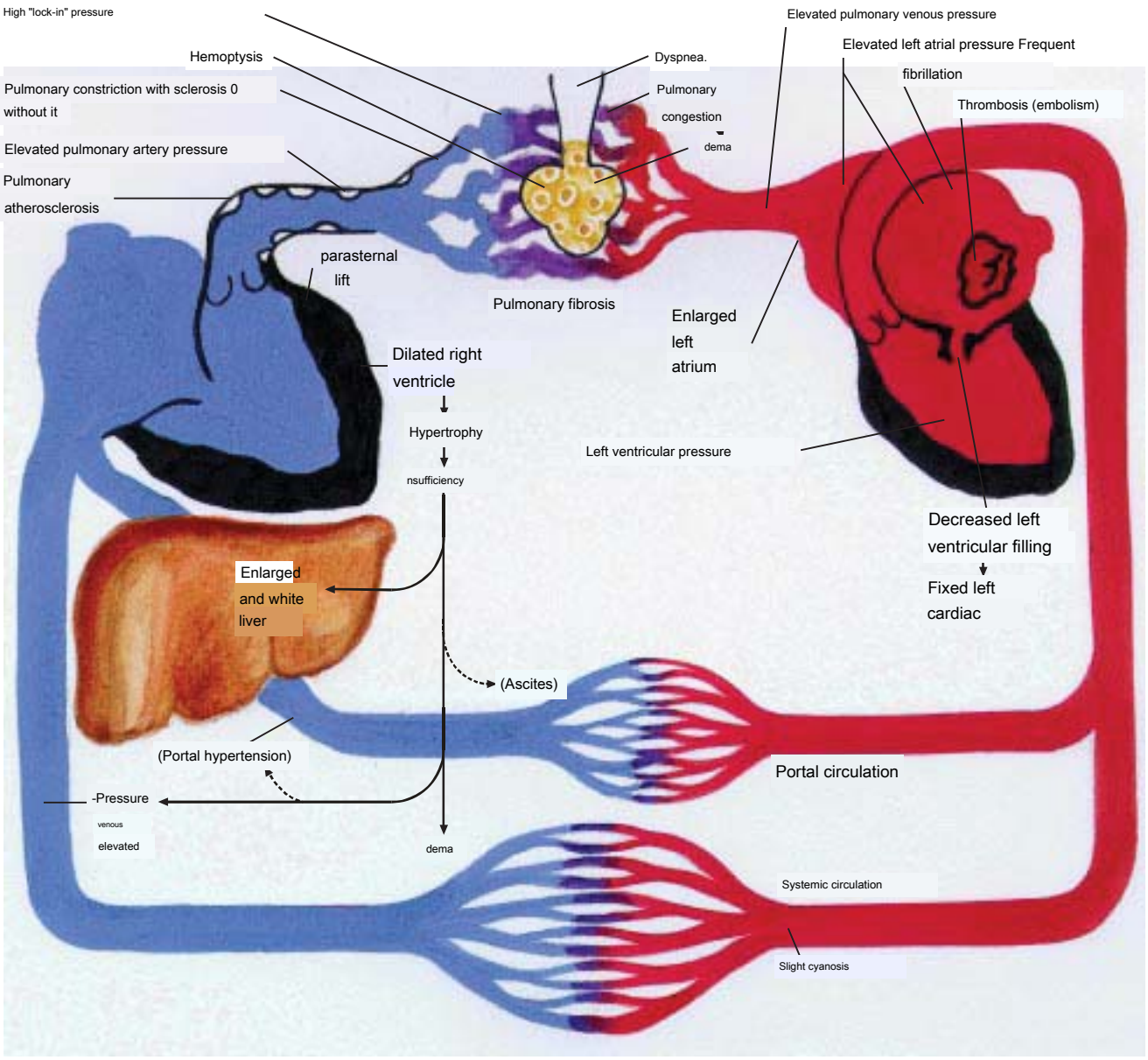


Fig. 45.7 Mitral stenosis.

High blood pressure and aortic stenosis increase this regurgitation. Secondarily, there is dilation of the left atrium and an increase in pressure that is transmitted retrogradely to the pulmonary veins, creating circulatory stasis in the lung.

It also has an anterograde impact on the left ventricle, which has to deal with the regurgitation of atrial blood and overcome the aortic sigmoid ducts.

Syndromography or positive diagnosis

Clinical picture

1. Dyspnea on exertion as an early symptom: palpitations.
2. Mitral facies with distal cyanosis (Lasègue blue cardiac).
3. Intense holosystolic murmur like a jet of steam at the tip, radiating to the axilla and the base of the left lung.
4. Shock of the tip displaced outwards and very intense.
5. Small pulse (parvus) and rapid rise (celer).

Complementary exams

Radiology. Dilated left ventricle, as well as the left atrium.

Angiocardiography. Drop in minute volume of the left ventricle. Excessive volume of the left atrium. High systolic and low diastolic pressure.

Electrocardiogram. Signs of left ventricular hypertrophy and mitral P waves. Left axis. Overload-

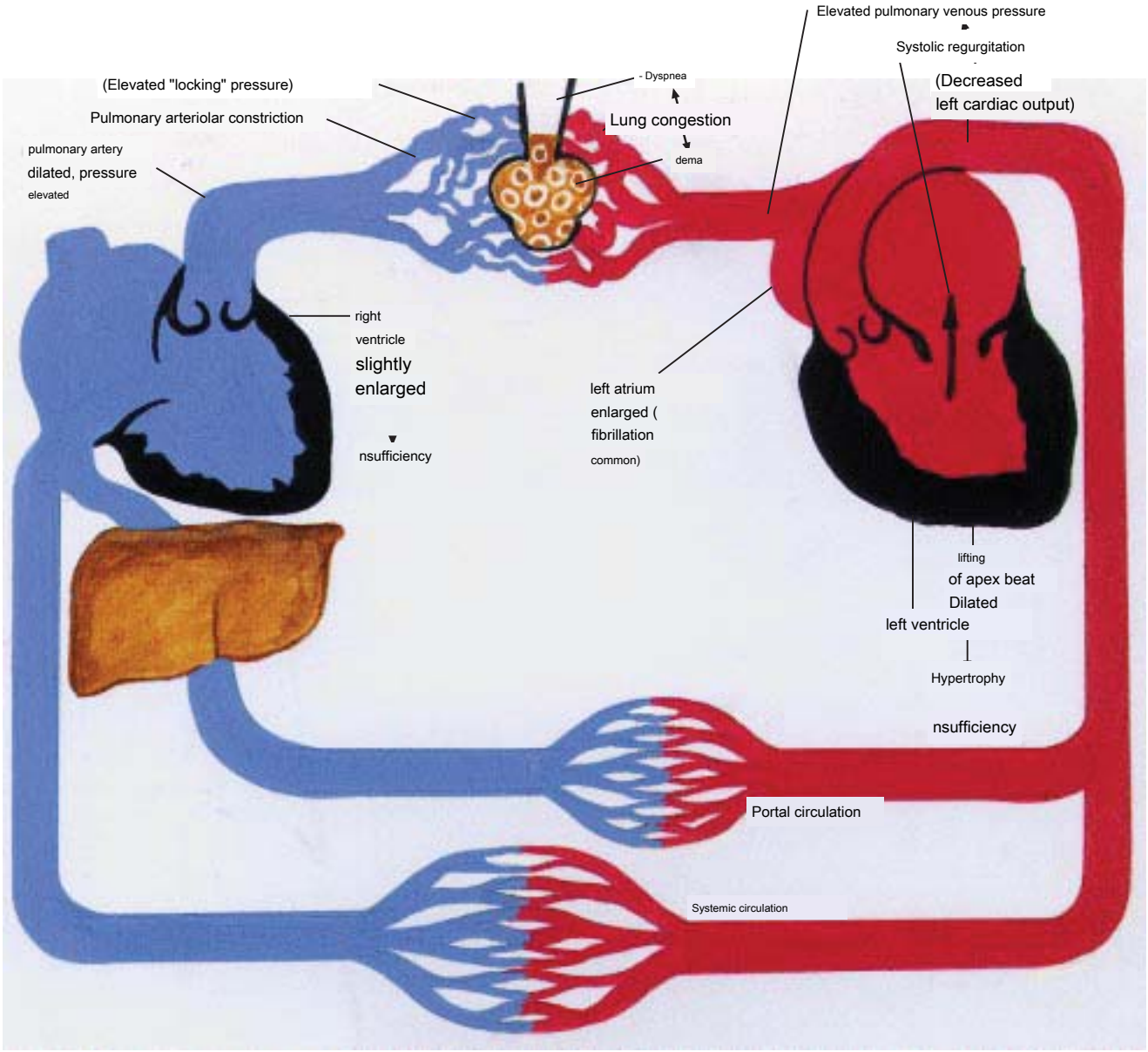


Fig. 45.8 Mitral regurgitation.

diastolic pressure of the left ventricle with growth of the left atrium.

Etiology

Mitral insufficiency is more common in acute articular rheumatism (90-95%), in the destruction of a traumatic muscle pillar or due to myocardial infarction, and in bacterial endocarditis. Dilations of the left ventricle give rise to functional mitral insufficiency. Mitral regurgitation can be congenital.

MITRAL LVULA LAV PROLAPSE SYNDROME

Nowadays, mitral valve prolapse has been reported with increasing frequency, which will be studied here because it is often confused with the mitral syndromes mentioned above.

Concept

Described a few decades ago, it is one of the most frequent valve syndromes and for many authors the main cause of mitral insufficiency or regurgitation. It is caused by the waving, hernia or prolapse of one or both valves or leaflets, usually the posterior one, towards the left atrium during systole, the starting point of an alteration of the structure and therefore of the valves and tendine cords of not well established origin.

It presents a clinicopathological spectrum that ranges from mild prolapse without regurgitation to valve prolapse with mitral regurgitation that arises when the leaflet edges do not coapt.

Syndromog nesis or pathophysiology

Myxomatous degenerative changes occur in the valvular apparatus, particularly in the leaflets, which are characterized by the abnormal increase in the mucous layer that invades the fibrous supporting layer of the cusps. These weaken and then prolapse or bulge during systole in the left atrium. The lengthening of the weakened chordae tendineae must contribute to this. Inflammatory changes may also play a role.

Usually a myxomatous mitral valve, in the absence of complications, is sufficient. The increase in tension in the chordae tendineae and the sudden deceleration of blood flow under the prolapsed mitral leaflet during systole cause the systolic click.

For failure to occur with regurgitation, inadequate coaptation of the mitral valve during systole is necessary; thinning of the cusps, lengthening of the chordae tendineae, excessive valve tissue and, sometimes, dilation of the mitral annulus. The severity of mitral regurgitation depends on the degree of prolapse.

Other pathophysiological mechanisms that appear to play a role in this syndrome are abnormalities of ventricular systolic constriction, myocardial ischemia, coronary spasm, and neurogenic mechanisms. It should not be confused with the so-called secondary prolapse of the mitral valve in which the leaflets are macroscopically and histologically normal, but the dysfunction of the valve, as well as other factors, prevent its adequate closure and cause prolapse of the leaflets, as observed in ischemia or infarction of papillary muscles or the adjacent myocardium.

Syndromography or positive diagnosis

Clinical picture

More than two thirds of the patients are women. There may be first-degree relatives with the syndrome.

High proportion of asymptomatic people.

Symptoms. Palpitations, fatigue, anxiety, atypical precordial pain (prolonged, does not occur with effort and is constrictive), laxity, fainting, vertigo and even syncope. Neurological attacks of transient amaurosis may appear.

Signs. Asthenic habit. Anomalies of body habitus with narrower anteroposterior thoracic diameters, straight back, excavated thorax, long limbs and hyperreflexic joints. Keeled thorax. There may be scoliosis or kyphosis or absence of thoracic kyphosis.

Hypomastia. Orthostatic hypotension may occur.

Auscultation. Typical signs and major diagnostic criteria are the mid-systolic click (although it can be proto or end-systolic) and the end-systolic murmur (although it can be mid-systolic and in 10% hol-systolic) or both.

The click is louder at the left sternal border or at the apex of the heart, while the murmur is more audible at the cardiac apex.

The standing position and the administration of amyl nitrite (maneuvers that reduce the volume of the left ventricle) cause the prolapse to occur earlier, and therefore, the click and murmur occur at the beginning of systole. On the contrary, the recumbent or squatting position (maneuvers that increase the volume of the left ventricle) cause the murmur and click to appear later in systole.

Complementary exams

Electrocardiogram. It may be normal or present non-specific changes in the ST segment and the T wave, especially inversion of the T waves in DIII and aVE. QT prolongation.

Chest x-ray. Normal or reduction of the anteroposterior diameter. Keel chest, normal heart size, without significant mitral insufficiency, presence of cardiomegaly.

Echocardiogram. Both M mode and B mode (the latter is superior in diagnosis) demonstrate systolic prolapse of the leaflets, if there are cord breaks and flaccid cusps.

Doppler ultrasound. To assess the presence and severity of mitral regurgitation.

Radionuclide scintigraphy. To calculate the severity of mitral regurgitation.

Cardiac catheterization. It may demonstrate prolapse of the leaflets or cusps. It allows mitral regurgitation to be calculated. Defines the coronary anatomy.

Etiology

- Mitral valve prolapse as a primary disease that is not related to another condition (sporadic or familial).

- Associated with other conditions, such as:

Inherited connective tissue disorders.

Marfan syndrome.

Osteogenesis imperfecta.

Pseudoxanthoma elasticum.

TRICUSPID STENOSIS FIG.

Concept

It consists of the narrowness of the right atrioventricular valve, which makes it difficult for blood to pass from the right atrium to the right ventricle.

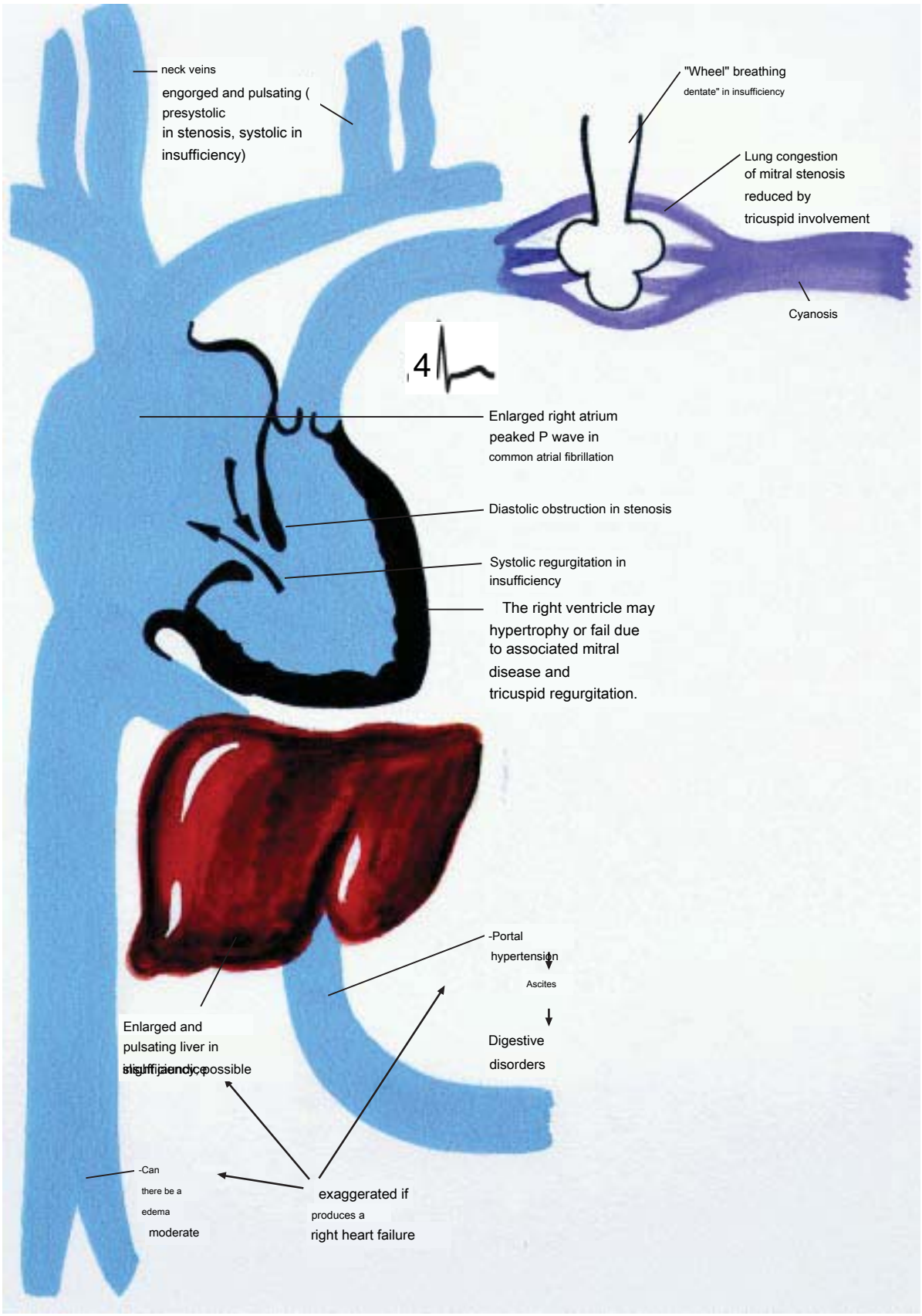


Fig. 45.9 Tricuspid stenosis and tricuspid insufficiency.

Syndromog nesis or pathophysiology

The difficulty in passing blood from the right atrium to the right ventricle causes right atrial stasis with hypertrophy of said atrium. This stasis will later extend to the venous territory of its tributaries, manifesting itself by venous engorgement of the neck with increased central venous pressure (greater than 12 cm of water) and also by painful congestive hepatomegaly, ascites, edema in the lower limbs and hepatojugular reflux (see tricuspid insufficiency). The right ventricle will have a decreased volume and thickness, as it receives little blood volume.

Syndromography or positive diagnosis

Clinical picture

1. Moderate dyspnea, with little response to cardiotonics.
2. Enormous venous engorgement of the neck.
3. Presystolic murmur with tricuspid opening click.
4. Constant painful congestive hepatomegaly with ascites. Edema in lower limbs less frequently.

Complementary exams

Radiology. The contrast between the large right atrium and the clear lung parenchyma is striking. Sometimes even the arch of the azygos vein can be seen.
Normal right ventricle. The importance of contrast-enhanced esophagus is emphasized, because its deviation to the left favors right atrial dilation and tricuspid stenosis.

Phonocardiogram. Presystolic murmur with tricuspid opening click. It is not accompanied by reinforcement of the second lung sound, as occurs in mitral stenosis.

Angiocardiography and cardiac catheterization. Large right atrial cavity that empties slowly. Contrast reflux into the azygos, superior cava, and suprahepatic veins. Weak opacification of the right ventricle and pulmonary artery. The pressure in the right atrium is naturally higher than in the right ventricle, at the end of diastole (normally they are equal).

Electrocardiogram. Elevated P waves without signs of right ventricular hypertrophy.

Etiology

It is always organic (congenital, rheumatic or post-scarlet fever), but it is never functional. It can be solitary or associated with collagen diseases or argentophilic intestinal tumors. It is the worst tolerated atrioventricular valve disease.

TRICUSPID INSUFFICIENCY SEE FIG.

Concept

It is the lack of occlusion of the tricuspid orifice during ventricular systole, which allows blood to reflux into the right atrium.

Syndromog nesis or pathophysiology

Regardless of its etiology, when there is tricuspid regurgitation, the right ventricular systole sends part of its volume in the form of reflux towards the right atrium, which dilates secondarily and produces blood stasis in the vessels that reach it:
superior vena cava and inferior vena cava. This dilation of the right atrium can lead to atrial fibrillation. During diastole, the right ventricle will receive a greater volume of blood from the atrium and this fact will lead it first to dilation and then to hypertrophy, while the left ventricle will be smaller due to receiving less return volume.

On the other hand, hypertension in the superior vena cava will result in great engorgement of the internal and external jugular veins, with its characteristic systolic venous pulse in the neck, capable of elevating the sternocleidomastoid muscle; Venous hypertension in the territory of the inferior vena cava will cause congestive and painful hepatomegaly, characterized by a palpable and expansive systolic hepatic pulse. Finally, it will be accompanied by ascites and edema in the lower limbs. As we can see, this condition of the right heart is not exclusive to organic tricuspid insufficiency, but common to any cause that causes right heart failure (pulmonary and mitral stenosis, acute or chronic pulmonary hypertension, compression by mediastinal tumors and pericarditis with effusion or cardiac tamponade).

Syndromography or positive diagnosis

Clinical picture

1. Always moderate dyspnea and without nocturnal attacks of paroxysmal dyspnea or acute lung edema.
2. Venous engorgement of the neck, with systolic venous pulse and evident Rondot's hepatojugular reflux;
   This is reflected in a very high central venous pressure.
3. Systolic murmur in the tricuspid focus, which appears in the xiphoid region, with a harsh character and a high tone over the left edge of the sternum that spreads towards the tip and the right axilla. Sometimes it is accompanied by thrills.
4. Painful congestive hepatomegaly, which may be accompanied by ascites, edema in the lower limbs and hepatojugular reflux.

Complementary exams

Radiology. Great dilation of the right contour corresponding to a very visible atrium in the right anterior oblique position (RAO). Superior vena cava dilated and easily visible in a posteroanterior position, ascending and parallel to the vertebral column. Normal clarity of lung fields.

Cardiac catheterization. Right atrial hypertension (up to 20-25 mm Hg), with poor cardiac performance. This fact causes elevated central venous pressure.

Electrocardiogram. Right axial deviation and incomplete right bundle branch block. There may be signs of right ventricular hypertrophy.

Etiology

This insufficiency can be organic and rarely pure, rather associated with other organic valvular lesions;
It is almost always secondary to endocarditis. Functional failure is very common during right ventricular dilation.

AORTIC STENOSIS FIG.

Concept

It consists of a decrease in the caliber of the aorta, generally at the level of the sigmoid valves, due to lesions that are almost always inflammatory or degenerative, leaving behind hardening and fusion between the leaflets.

Syndromog nesis or pathophysiology

In ventricular systole, the aortic sigmoid orifice has a diameter of 3 cm. The stenotic lesion is not symptomatic until the caliber is reduced to less than 1 cm. As a consequence, the wall of the ventricle thickens enormously (concentric hypertrophy) and the capacity of the left ventricle is reduced, lengthening the ejection period. During systole, the pressure in the left ventricle rises well above the peripheral arterial pressure (and can reach 200 or 300 mm Hg), with blood projected out in an energetic and fine jet, which produces a true aspiration of blood from the coronary territory (Venturi effect).

Syndromography or positive diagnosis

Clinical picture

1. Dyspnea on medium and minor efforts.
2. Angina pectoris crisis in 34.8% of cases.
3. Sudden death and seizures in 20.6% of cases

s.o.s.

4. Porcelain facies (pale like Lasègue's white cardiacs).
5. Pulse tardus and parvus (slow and small).
6. Low maximum blood pressure, with reduced differential.
7. Mid-systolic murmur, elongated and raspy, which begins after the first sound and ends immediately before the second. Its greatest intensity is in the aortic focus. It radiates to the vessels of the neck and is accompanied by a thrill on palpation.

Complementary exams

Radiology. Post-stenotic dilation of the ascending portion of the arch of the aorta. The left ventricle shows its round tip, typical of concentric hypertrophy. The presence of calcifications in the valves is a decisive diagnostic element.

Angiocardiography. Moderate dilation of the left atrium, and evident and late dilation of the left ventricle, with retention of the contrast in the latter, due to the difficulty in its evacuation.

Cardiac catheterization. The ventricular systolic pressure is elevated (it can exceed 200 mm Hg) and there is a lengthening of the total duration of evacuation due to the prolonged phase of slow ventricular emptying produced by aortic stenosis.

Electrocardiogram. It indicates left ventricular hypertrophy with systolic overload.

Etiology

It predominates in males and is usually of endocardial, rheumatic (67% of cases), congenital or arteriosclerotic origin.

AORTIC INSUFFICIENCY FIG.

Concept

It is the inability of the sigmoid valves to keep the aortic orifice closed during diastole.

Syndromog nesis or pathophysiology

During diastole, the blood that refluxes into the left ventricle from the aorta is added to that which normally comes from the left atrium. The left ventricle fills excessively, consequently it is forced to perform greater systolic work, which causes its hypertrophy and dilation. This causes an increase in systolic blood pressure with a drop in diastolic blood pressure, which explains many symptoms observed in this syndrome.

Syndromography or positive diagnosis

Clinical picture

1. Dyspnea evolving from exertional dyspnea to paroxysmal dyspnea and orthopnea. When the insufficiency

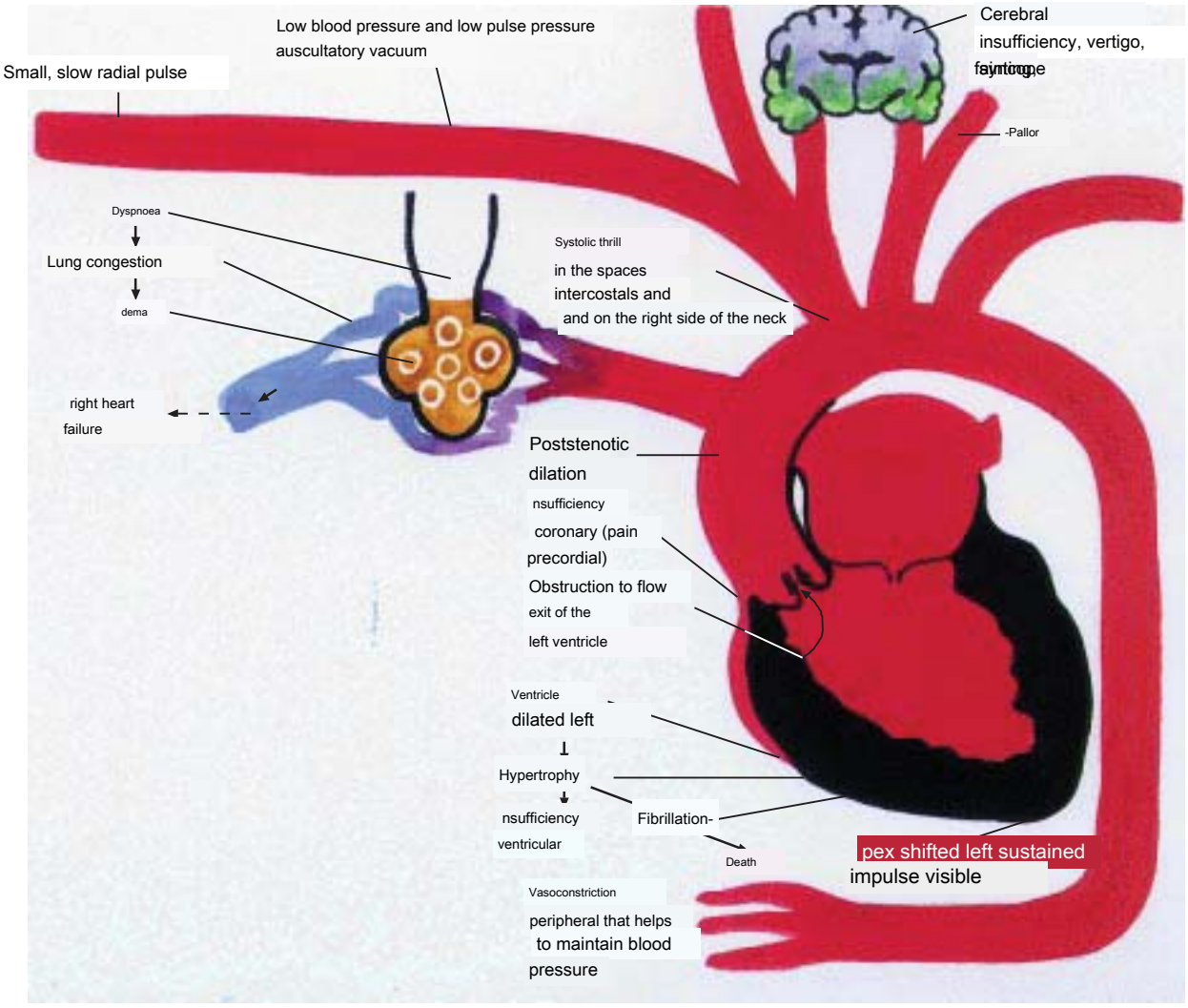


Fig. 45.10 Aortic stenosis.

The right heart rate is added to the left signs, so the dyspnea improves.

2. Pale facies ("white cardiacs" of Lasègue).

3. Cardiovascular manifestations:

a) High maximum blood pressure with very low diastolic and large differential. Hard, jumping pulse (Corrigan's pulse). At the femoral level, Duroziez's double crural murmur was heard.

b) Soft, aspirational, high-pitched diastolic murmur heard as a whispered R, starting with the second sound. It is best heard along the left edge of the sternum with the patient leaning forward and in post-expiratory apnea.

c) Strong systolic murmur called accompanying because the reflux of blood in protodiastole produces an increase in the volume of blood in the left ventricle, which gives rise to dilation and hypertrophy by creating a relative stenosis of the aortic sigmoid at the time of systole, which which, together with the irregularities of the valve, gives rise to this murmur.

d) Diastolic dragging or rumbling without thrill (presystolic), reminiscent of mitral stenosis, known as Austin Flint murmur, caused because a relative mitral stenosis occurs due to dilation of the left ventricle, or because the healthy mitral leaflets are very close at the time of atrial systole.

Complementary exams

Radiology. Left ventricular hypertrophy and dilation of the ascending aorta, aortic button and well-marked descending aorta. Left ventricle and aortic arch hyperkinetic on fluoroscopy.

Phonocardiogram. Intense, decreasing holodiastolic murmur that begins with the second heart sound.

catheterization. The systolic pressure in the left ventricle and aorta, as well as the diastolic pressure, is somewhat elevated;

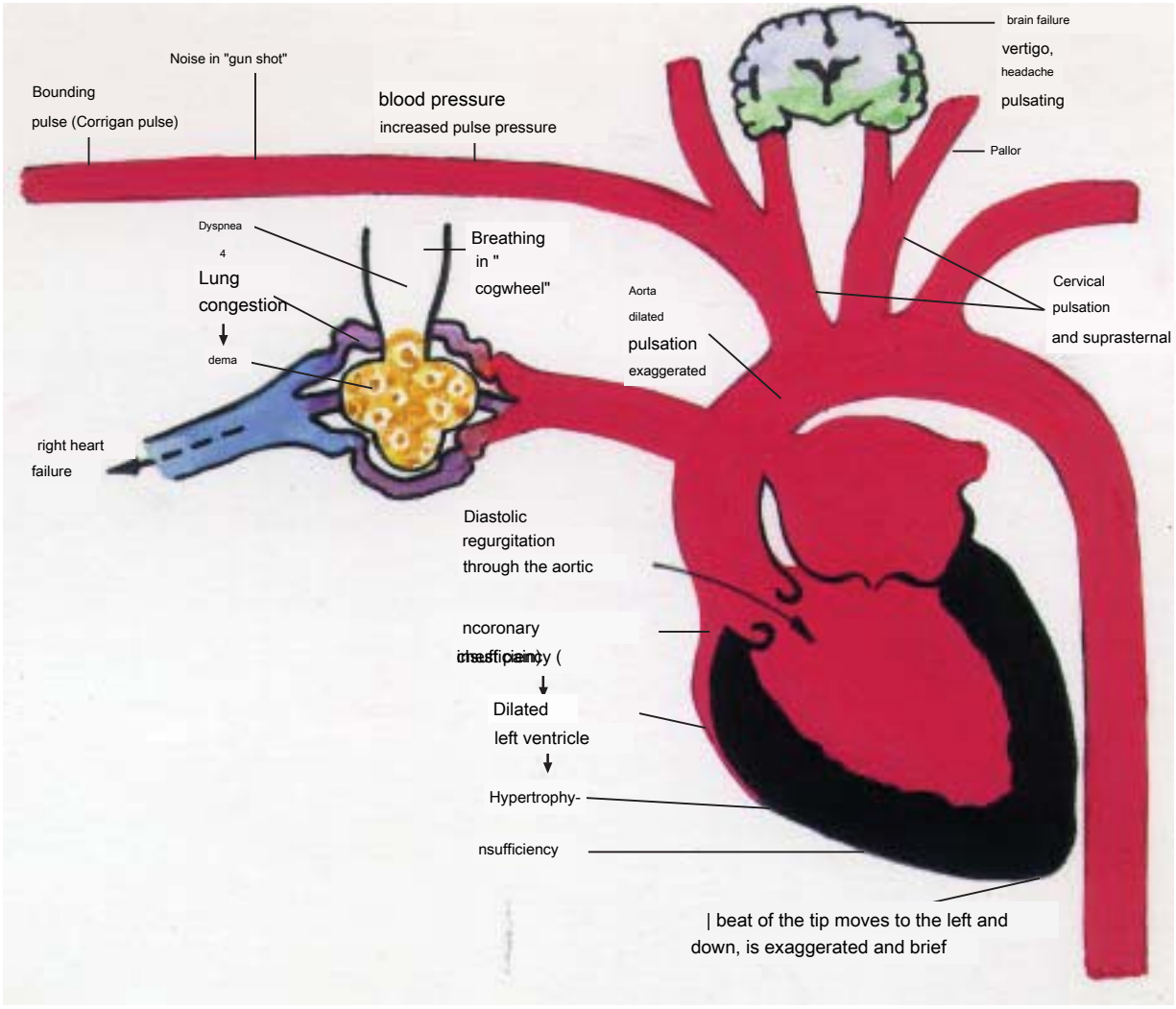


Fig. 45.11 Aortic insufficiency.

the latter due to blood reflux that occurs from the aorta to the left ventricle.

Electrocardiogram. Electrical axis on the left with left ventricular hypertrophy and diastolic overload morphology.

Etiology

Generally of rheumatic or luetic origin and less frequently secondary to endocarditis, atheromatosis, arterial hypertension and dissecting aneurysm of the aorta.

PULMONARY STENOSIS

Concept

It consists of a decrease in the caliber of the pulmonary artery at the level of the sigmoid valves due to inflammatory or congenital lesions.

Syndromog nesis or pathophysiology

The difficulty of blood passing through this stenotic pulmonary valve determines a manifest dilation. tion and hypertrophy of the right ventricle, with a reduction in the volume of blood that reaches the pulmonary fields and consequent alteration of hematosis, which is reduced.

Syndromography or positive diagnosis

Clinical picture

Exertional dyspnea with cyanosis when hematosis is deficient and right heart failure is marked.

Complementary exams

Radiology. Bulging of the left pulmonary arch. Increase in volume of the right chambers with elevation and displacement of the tip of the heart to the left (Swedish shoe appearance).

Cardiac catheterization. Hypertension of the right chambers and especially of the ventricle, with normotension in the remaining chambers. Hypotension in the pulmonary artery.

Electrocardiogram. Right axial deviation. Lung waves and right ventricular systolic overload

cha.

Etiology

It can be acquired, of rheumatic origin or congenital.

The congenital form rarely occurs in isolation, but is associated with other anomalies: atrial septal defect, ventricular septal defect, trilogy or tetralogy of Fallot.

LUNG FAILURE

Concept

Inability of the pulmonary sigmoids to keep the lung orifice closed during diastole.

Syndromog nesis or pathophysiology

The insufficiency of the pulmonary sigmoid valve allows a normal systole, but there is a strong reflux of blood in diastole that engorges and overloads the right ventricle, which dilates and hypertrophies in the face of this greater blood volume. Progressive claudication of the right ventricle would give rise to stasis above it, dilating the right atrium and transmitting this hypertension to the tributary territory of the superior and inferior cavas, with its corresponding manifestations.

Syndromography or positive diagnosis

Clinical picture

1. Little dyspnea.
2. Moderate neck venous engorgement.
3. Discreet diastolic murmur without thrill, spreading along the left edge of the sternum (Graham-Steell murmur). The second noise may be reinforced or duplicated.
4. Constant painful congestive hepatomegaly.
5. Very high central venous pressure when right heart failure is present.

Complementary exams

Radiology. Increase of the right cavities; prominent left middle arch (pulmonary arch). In the fluoroscope, a hilar dance can be observed, as a result of a large systolic volume alternating with diastolic reflux.

Phonocardiogram. Decreasing, high-frequency diastolic murmur that begins with the pulmonary component of the second sound.

Electrocardiogram. Electrical axis on the right, with conduction disorders or right bundle branch block. Signs of ventricular hypertrophy and right overload.

Etiology

It is usually functional: it is secondary when it is caused by pulmonary hypertension, or primitive when it is caused by: mitral stenosis, chronic cor pulmonale, atrial septal defect, etc. It is rarely organic.

PERIC RDICO SYNDROME

Pericardial syndrome is made up of clinical manifestations resulting from irritation, invasion or injury to the membranes that surround the heart (pericardium), both at the level of the visceral and parietal leaf, or both.

Clinically we can distinguish two essential types of pericardial conditions:

- Acute pericarditis.
- Constrictive pericarditis.

ACUTE PERICARDITIS

They constitute an inflammatory process, almost always caused by a bacterial infection or by a systemic disease such as rheumatic fever. Pericarditis may be the only cardiac lesion or may be associated with myocarditis, endocarditis, or both (Fig. 45.12).

Syndromog nesis or pathophysiology

Inflammation of the pericardium gives rise to an effusion in the pericardial cavity, which can be fibrinous, serofibrinous, hemorrhagic or purulent. On other occasions the pericardium reacts in such a way that it produces adhesive pericarditis.

Syndromography or positive diagnosis

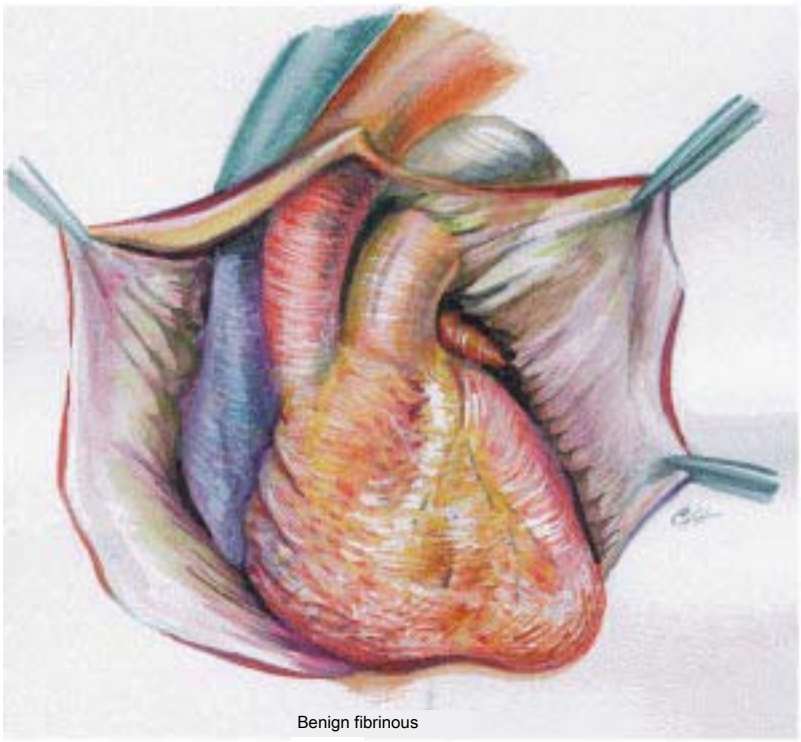
Clinical picture

Subjective symptoms

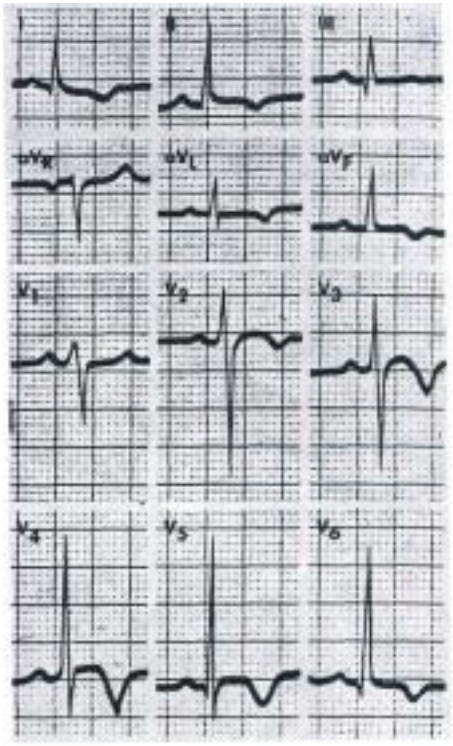
The main ones are:

- Precordial pain or discomfort.
- Dyspnea and other symptoms resulting from compression of the heart and neighboring thoracic structures.
- General symptoms.

Chest pain or discomfort. It may be absent or very high-pitched and intense, or dull and vague. It is located in the precordium, and can radiate to the left shoulder,

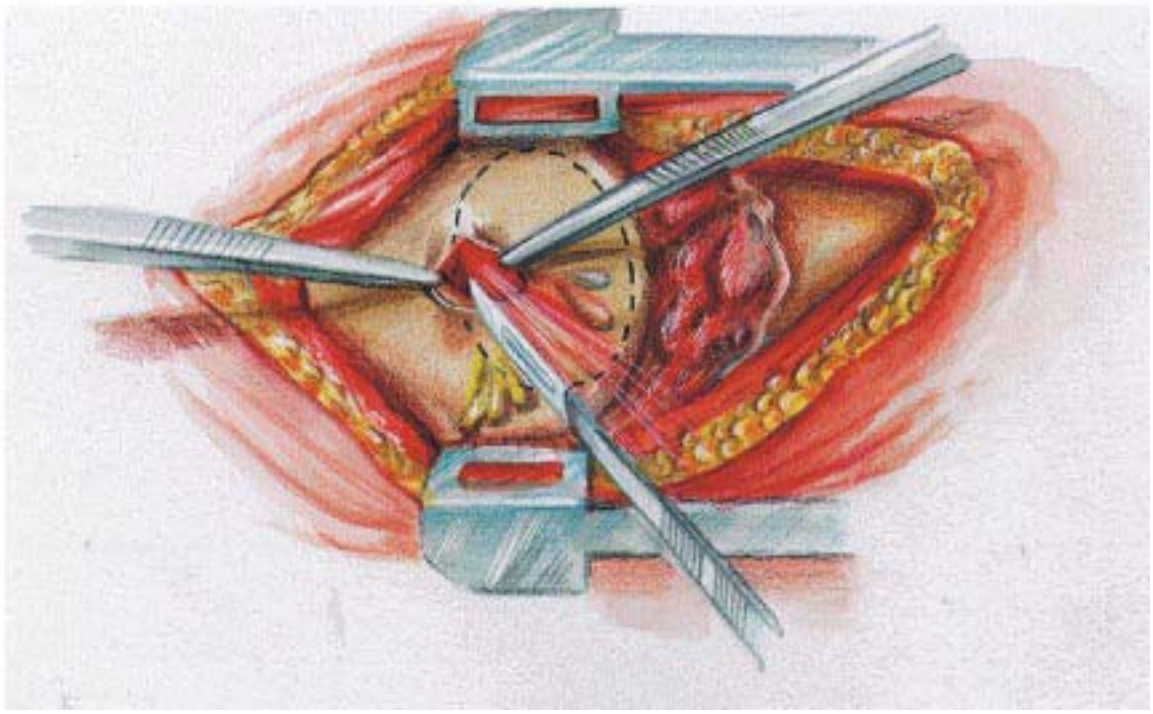


Benign fibrinous pericarditis



Characteristic CG changes

T wave inversion in all leads except aVR and V, isoelectric in D



Pericardial effusion, a pleuropericardial window is created and a biopsy is performed, after making the incision in the left fifth intercostal space

Fig. 45.12 Acute pericarditis.

neck and arm, to the epigastric region and to the left scapular region, often simulating the pain of coronary occlusion. Sometimes, when the pain is located only in the abdominal region, it can be diagnosed equi-
vOccasionally an abdominal condition. It is frequently exacerbated by deep inspiration, chest wall movements, and coughing.

Dyspnea and other symptoms resulting from compression of the heart and neighboring thoracic structures. Dyspnea is usually the earliest, most frequent and most prominent symptom in cases of pericarditis with effusion, which requires the orthopneic position and other attitudes such as " Mohammedan prayer." Dyspnea is largely the result of mechanical compression of the bronchi or lungs and in cases of large effusions it is due to the decrease in vital capacity caused by a reduction in the available thoracic space.

Other symptoms that may appear are persistent dry cough, dysphonia or dysphagia due to compression of the trachea, bronchi, lungs and esophagus, which occur in cases with large spills, such as tuberculous pericarditis.

General symptoms. They may be due to pericardial infection or inflammation or to the systemic disease of which pericarditis is a part. Depending on the etiology of pericarditis, the following may appear: fever, sweating, chills, fatigue, weakness (asthenia), weight loss, anxiety, depression, delirium, etc.

Objective symptoms

The main ones are:

Physical signs in the chest.

- Manifestations produced by altered hemodynamics.

- General manifestations.

Physical signs in the chest. They are produced by the inflammation of the pericardium and the presence of the effusion. They are:

a) Pericardial friction or fremitus. Obtaining by palpation or by auscultation of a pericardial rub is conclusive evidence of acute pericarditis. It is frequently audible to the left of the lowest portion of the sternum, in a small, well-defined area. Its location can vary with changes in the patient's position, being heard better if the patient leans forward and if moderate pressure is exerted on the chest wall with the bell of the stethoscope. We must here repeat that pericardial rub is obtainable in dry pericarditis or in pericarditis with effusion, before it appears or after its resorption.

b) Transverse increase in cardiac submatidity at the base. The left border of the cardiac submatidity in The second and third left intercostal spaces are noticeably displaced to the left, resulting in a widening of the submatidity area at this height, mainly when the patient is in a lying position, decreasing if the patient sits, since the effusion descends due to the action of gravity.

c) Generalized increase in cardiac submatescence. When there is a large volume pericardial effusion, percussion of the precordial area reveals a generalized increase in cardiac submatidity, which takes on a globular shape if the patient is lying down.

d) Sign of Ewarto sign of Pins. In cases of large pericardial effusion, especially in children, there is frequently an area of submatidity below the angle of the left scapula. In this area of dullness we can obtain bronchial breathing and usually bronchophony or egophony. This sign is due to the compression that the pericardial effusion exerts on the base of the left lung.

e) Other physical signs. Less specific, less constant or perhaps of dubious value, are those signs that we can also obtain during the physical examination of the precordial area, such as the absence or decrease of heartbeats that are normally visible or palpable; and heart tones, which may be muffled or distant when the patient is in a lying position.

Manifestations produced by altered hemodynamics. They are due to cardiac tamponade or compression caused by high intrapericardial pressure. Cardiac tamponade will be described in a separate syndrome considering the urgency in the therapy that it brings with it.

General manifestations. The patient may be anxious, restless, cyanotic or pale and assuming a characteristic attitude; He sits up in bed and leans forward, often adopting the "Mohammedan prayer" position.

Complementary exams

Radiological study. Serial radiological study of the chest, frequently performed in the standing and semi-sitting positions, is of great value in confirming the diagnosis of pericardial effusion. The most prominent signs of the radiological examination are: blurring of the reference points of the different cardiac chambers, resulting in a smoothing of the cardiac edges, which produces a silhouette that is compared to that of a large inverted cup: and the supracardiac vascular pedicle appears shortened and widened.

Radioscopic study. A marked decrease or complete absence of visible cardiac pulsations is observed.

Angiocardiography. Through it we obtain a characteristic shadow that shows the opacification of the cardiac chambers by the contrast medium, this shadow is kept at a distance from the global contour of the radiological image.

Electrocardiogram. It reveals characteristic changes of ST and T that we can summarize as:

a) Elevation of the ST segment in the three standard leads in leads I and II, in leads II and III or in lead I. There is no reciprocal ST depression in the standard lead in which it is not elevated, which is characteristic of myocardial infarction.

b) In the precordial leads there may also be elevation of the ST segment or inversion of the T waves.

c) Changes in the T wave, which is usually positive in standard leads, are related to the evolution of the process, and its progressive flattening can be observed during the regression of ST to the isoelectric line. Weeks after the beginning of the process the T wave can become inverted. This reversal may last longer than ST changes, but usually returns to normal within 2-3 weeks.

d) We will also find a decrease in the voltage of the QRS complexes.

Etiology

Acute pericarditis is classified according to the bacterial agent that causes it, or according to the disease of which it is a part or complication, and thus we have: rheumatic pericarditis, tuberculous pericarditis, neurococcal bacterial pericarditis, streptococcal, uremic pericarditis, etc.

CONSTRICTIVE PERICARDITIS

Concept

They consist of a dense, fibrous and inextensible thickening of the pericardium.

Syndromog nesis or pathophysiology

Usually of uncertain etiology, although sometimes they may be due to tuberculous or bacterial origin. The clinical manifestations of constrictive pericarditis are motivated by hemodynamic disorders, a consequence of chronic compression of the heart that interferes with its diastolic relaxation and filling, and as a result gives systemic venous hypertension and a decrease in stroke volume. This is a true adiastole (Fig. 45.13).

Syndromography or positive diagnosis

Clinical picture

The general clinical picture is reminiscent of very marked right heart failure, with early ascites.

The patient consults more frequently due to increased abdominal volume and dyspnea on exertion. The first is due to the accumulation of ascitic fluid, which can be associated with various digestive symptoms such as anorexia, fullness and pain. Dyspnea occurs more frequently during exertion; It can also appear at rest.

In the physical examination we can see:

1. Cyanosis. Light or moderate.

2. Paradoxical pulse. Appears sometimes.

3. Blood pressure. It is usually low, with a decrease in the differential at the expense of the systolic, fluctuating between 15 and 25 mm Hg. Corresponding to pulsus paradoxus, blood pressure usually falls with inspiration.

4. Hepatoascitic manifestations (Pick syndrome).

They are directly related to peripheral venous hypertension and are those that dominate the clinical picture, which is characterized by:

a) Stasis hepatomegaly, always present, of variable but always important volume, of normal consistency at first and hard shortly afterwards, accompanied by frequent jugular engorgement and hepatojugular reflux.

b) Very frequent ascites, sometimes considerable, appears before edema in the lower limbs and is often accompanied by collateral venous circulation; It results in a deformation of the abdomen.

The puncture fluid is a predominantly lymphocytic transudate without Koch bacilli.

c) Edema of the lower limbs, moderate and sometimes absent, which contrasts with the importance of hepatomegaly and ascites.

5. Thorax. This region is characterized by:

a) Pulsations in the precordial region are minimal or may be absent, which means that the heart is said to be calm.

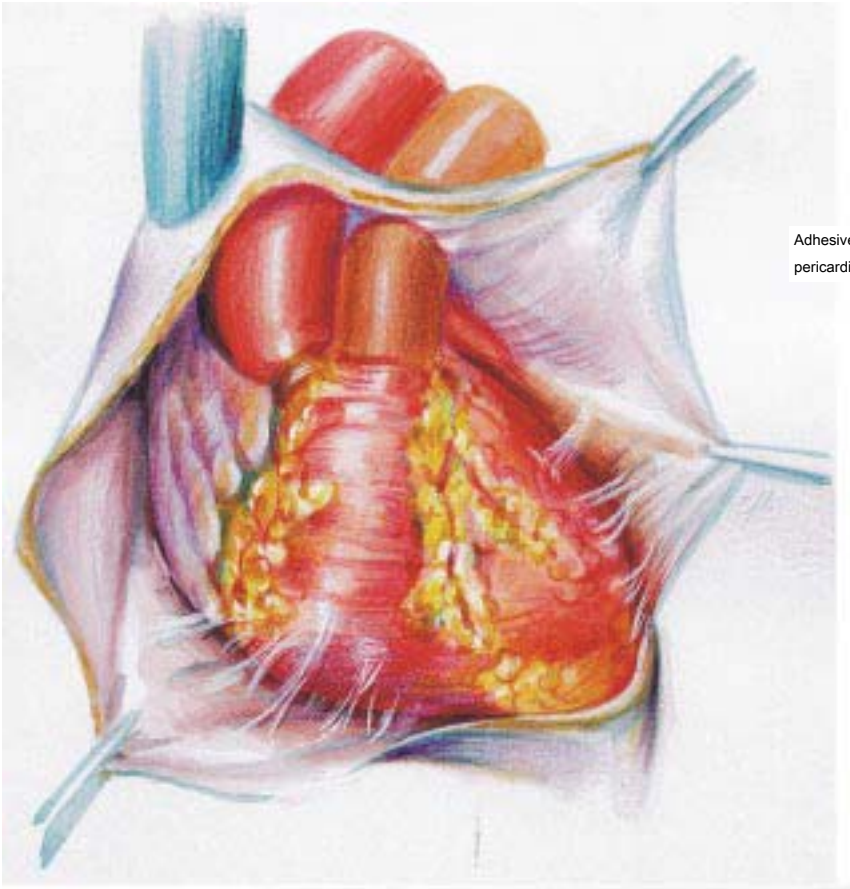
b) Percussion reveals a small heart or at least without significant enlargement.

c) Auscultation, we will not find evidence of valve injury. The noises will appear distant and muffled. The rhythm is usually normal sinus, there may be tachycardia.

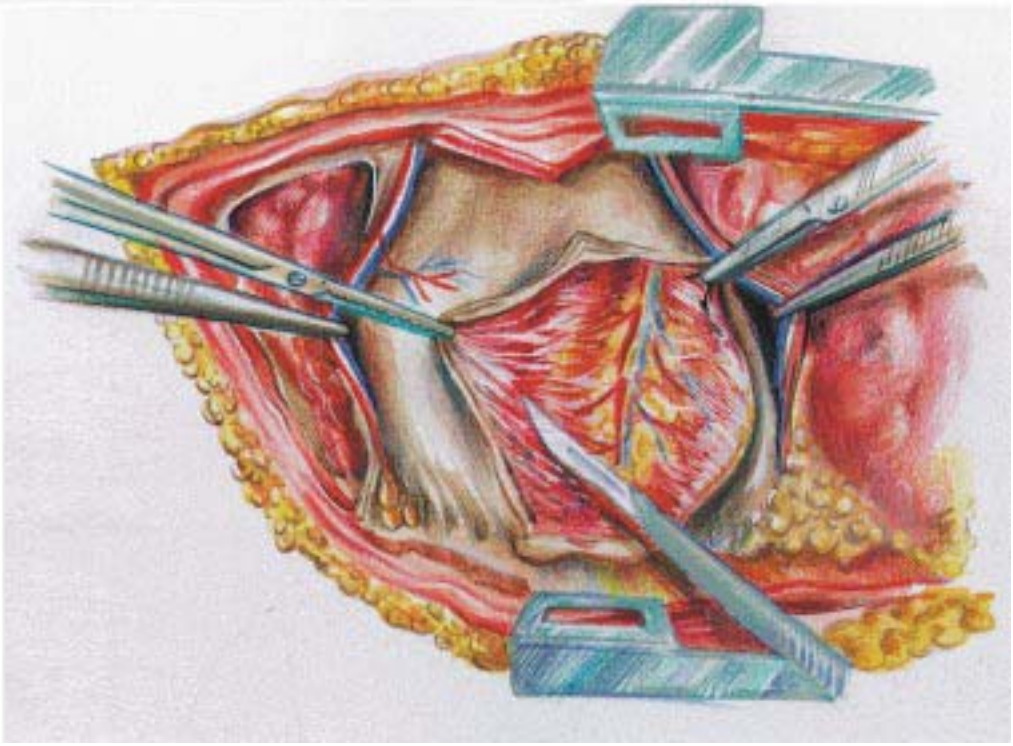
d) Upon examination of the lungs we usually find signs of pleural effusion.

Complementary exams

venous pressure. It is markedly and persistently elevated, with figures between 25 and 35 cm H,0. When pressure is exerted on the right hypochondrium, a marked elevation in venous pressure occurs.



Adhesive pericarditis



Excision of the constrictive pericardium with a transsternal incision preserves the phrenic nerves and accompanying

Fig. 45.13 Constrictive pericarditis.

Circulatory speed. It may be normal or the time may be prolonged due to venous stasis. The arm-to-tongue time is increased, from 20-40 s or more, but mainly at the expense of the arm-to-lung time.

Circulating blood volume. It may be increased by 30-45% above normal.

Stroke volume. It is diminished.

Vital capacity. Decreased between 36 and 68% of normal.

Radiological studies. The most frequent findings are:
calcifications of the pericardium; cardiac silhouette usually small; The right vascular shadow, which represents the superior vena cava, may be widened due to engorgement of the vessel; absence of congestion in the lung fields, which contrasts markedly with the clinical evidence of congestion of the systemic circulation. Pleural effusions and thickening are common.

Fluoroscopic studies. Decreased or absent heartbeats are observed.

Electrocardiogram. Typically, we find low voltage of the QRS complexes and flattening or inversion of T in leads I or II, or both. T alterations corresponding to the precordial leads and fixation of the electrical axis can be observed. Atrial fibrillation has been reported in a large number of cases.

Cardiac catheterization. It is essential not only to confirm the diagnosis, but to know before the intervention the degree of constriction of each ventricle.

All the pressures of the small circulation are uniformly increased at the expense, above all, of the diastolic pressures: from the pulmonary capillary pressure (which usually does not exceed 20 mm Hg on average) to the right atrial pressure (which can reach or exceed 20 mm Hg).

Mean right ventricular pressure and diastolic pressure are increased.

Catheterization of the left ventricle may show alterations identical to those of the right ventricle.

Etiology

Tuberculosis is responsible for three-quarters of the cases.

Bacterial pericarditis or pericardial hemorrhages rarely leave sequelae.

CARDIAC TAPONDING SYNDROME FIG.

CONCEPT

Cardiac compression or tamponade is said to exist when intrapericardial pressure increases to the degree of prevent ventricular filling, therefore modifying myocardial contraction. This intrapericardial pressure can increase for different reasons.

SYNDROMOGENESIS OR PHYSIOPATHOLOGY

Once the effusion exceeds the distension limit of the pericardium, the additional increase in fluid produces a severe limitation of the diastolic expansion of the cardiac cavities that affects venous return due to compression of the right atrium; This compression causes regurgitation, venous stasis, and increased central venous pressure.

The compression of both ventricles equally gives rise to an increase in pressures at the end of diastole and a decrease in volumes in that phase, which results in a drop in stroke volume and cardiac output.

The increase in pericardial effusion will increase these phenomena, which cannot be compensated by tachycardia, since it would cause even greater shortening of the diastolic full period, decrease in aortic pressure and increase in compression of the myocardium and coronary vessels, the latter causing coronary insufficiency. As heart failure increases and cardiac output decreases, sooner or later, total circulatory collapse occurs, that is, severe cardiogenic shock.

SYNDROMOGRAPHY OR POSITIVE DIAGNOSIS

It will be done through anamnestic data (history of pericarditis, systemic disease, tuberculosis, kidney disease or trauma) and physical examination.

clinical picture

1. Sometimes there is a comatose state or deep shock.
2. On other occasions, patients appear very restless, pale, cyanotic, sitting or leaning.
   two forward.
3. There is arterial hypotension with a decrease in differential pressure and pulse pressure.
4. Venous distention is significant, with marked jugular engorgement.
5. The pulse is rapid and weak, there is frequently a paradoxical
6. There is an increase in venous pressure, which can be seen due to the engorgement of these vessels and because they do not collapse during inspiration.
   The most common triad of cardiac tamponade is: venous hypertension, arterial hypotension, and an enlarged and still heart.
7. Rales are not seen in the lung fields.
8. Percussion and palpation of the heart do not reveal data of interest.

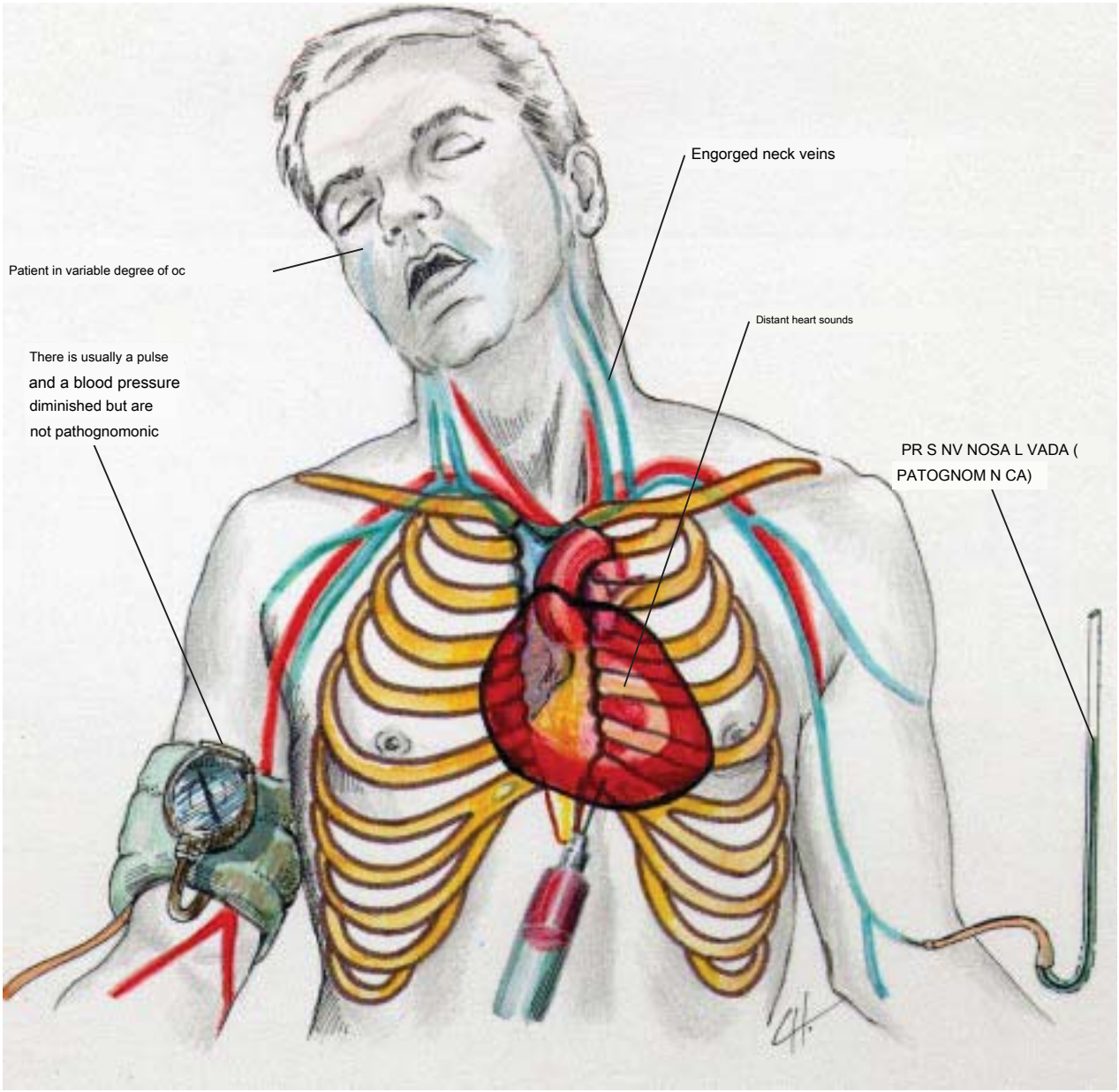

Engorged neck veins
Patient in variable degree of oc
Distant heart sounds
There is usually a pulse
and a blood pressure
diminished but are
not pathognomonic
PR S NV NOSA L VADA (
PATOGNOM N CA)

Fig. 45.14 Cardiac tamponade.

9. Cardiac auscultation shows a decrease in the intensity of heart sounds.
10. Hepatomegaly is painful, hepatojugular reflux is present.
11. There may be ascites, rarely peripheral edema.
12. In cases caused by trauma, other signs typical of this are associated.

Complementary exams

They do not provide useful data.

Pericardiocentesis. It is a mandatory and urgent procedure when there is a suspicion of blockage. His technique has already been described. 50-100 mL of liquid must be extracted, ob-

preserve its appearance and color and send it to the laboratory where the culture and cytological examination will be performed, which will help us in the etiological diagnosis of the syndrome. If the fluid is hemorrhagic due to trauma, surgical treatment should be considered.

ETIOLOGY

Inflammatory causes accompanied by massive exudation: tuberculous pericarditis; pyogenic pericarditis to other germs; neoplastic pericarditis; pericarditis in rheumatism and disseminated lupus erythematosus, and hemorrhagic pericarditis, which can be traumatic, due to excessive administration of anticoagulants, due to myocardial rupture due to infarction.

or by rupture of the aorta in its intrapericardial portion.

VENOUS IPERTENSION SYNDROME

CONCEPT AND PATHOPHYSIOLOGY

The venous system is responsible for collecting blood distributed in the tissues through the arteries and capillaries and leading it back to the heart.

The circulation of blood within the venous system is carried out, as in the arterial system, by the pressure differences between two adjacent points, going from the site of highest pressure to the site of lowest pressure.

There are three factors that determine venous tension:

1. The vis a tergo (vis: force; tergo: behind) that is, the rest of the cardiac impulse.
2. The expression of the veins by the contraction of the muscles.
3. Thoracic aspiration.

The driving action of these three factors and the resistance that the heart opposes to its filling, determine the existence of a certain pressure, which under normal conditions is around 100 mm H,0.

The increase in this pressure constitutes venous hypertension that can be generalized or localized to a certain sector and produced by various conditions as we will see later.

MEASUREMENT OF VENOUS TENSION

Venous pressure is measured in the superficial veins and given the low magnitude of its values, these are expressed in millimeters or centimeters of water, instead of millimeters or centimeters of mercury as in blood pressure.

Indirectly we can appreciate venous hypertension by observing the jugular veins or those on the back of the hand. If the jugular veins appear engorged when the subject is standing or sitting or if the veins on the back of the hand remain full when it is placed on a plane higher than that of the heart, we can assure that venous hypertension exists.

Normally the engorgement of the veins on the back of the hand decreases as it rises, to disappear when it reaches the phlebostatic level (height of the heart).

Instrumental methods (more accurate) measure venous tension after puncture of a superficial vein with a needle of a certain caliber (around 1 mm). The needle is connected to an aneroid, or better yet water, manometer, which consists of a graduated glass tube containing an anticoagulant solution.

The venous pressure measurement should be carried out with the subject in a horizontal position and, if possible, with the head and both arms resting on the plane of the bed.

It is essential that the zero of the manometer is in the same plane as the heart and that there is no compression above the site where the puncture is performed. The elbow vein is the one usually chosen for determination.

The normal value varies from person to person even under normal conditions. In general terms, it can be said that its normal value is 80-110 mm H,0. Central venous pressure can also be measured and is very useful, but requires the introduction of a polyethylene catheter into the cava.

CLASSIFICATION

Venous hypertension can be: localized or generalized.

localized venous hypertension

Localized venous hypertension occurs due to obstruction of a vein (thrombosis or compression), and causes edema and cyanosis of the corresponding territory. This hypertension exceeds 300 mm H,0.

In arteriovenous aneurysm, venous hypertension also occurs, but only in its vicinity, due to the direct passage of arterial blood to the venous system.

There are two types of localized venous hypertension:
Venous hypertension in the territory of the superior vena cava and venous hypertension in the territory of the inferior vena cava.

Venous hypertension in the territory of the superior vena cava

Compression or obstruction of the superior vena cava results in venous stasis in all its tributary veins. As a consequence of cephalic stasis, the patient may experience headaches, dizziness and ringing in the ears, and epistaxis and esophageal and tracheal hemorrhages may occur.

The most important signs of compression of the superior vena cava are: cyanosis and edema of the upper half of the body, venous engorgement, and the development of collateral circulation. We have already studied these signs in generalities.

Etiology

Among the etiological factors we can point out, first of all: mediastinal neoplasms, lymphomas, mediastinal lymph node metastases and lymphoid leukemia, which represent 50% of these factors.

Secondly, aneurysms of the ascending aorta that compress the superior cava (30%).

Finally, fibromediastinum, the open aortic aneurysm in the superior cava due to a small perforation, or thrombosis of the cava, which occurs exceptionally.

It is clear that in the generalized venous hypertension that we already studied there are manifestations of venous hypertension of the superior cava.

Venous hypertension of the superior cava is likely to hinder the emptying of the azygos major vein, which receives a large part of the venous blood from the right pleura and a smaller proportion of that which comes from the left pleura through the azygos minor, so hydrothorax may occur. We have already studied the different varieties that it can take depending on the location of the obstruction in relation to the azygos major vein.

Venous hypertension in the territory of the inferior vena cava

It determines venous stasis in the corresponding territory, which results in visceral stasis, the most typical examples of which are congestive hepatomegaly, serous effusions, and edema, especially in the lower limbs.

In generalized venous hypertension, the rise in blood pressure is greater in the lower territory than in the upper territory, probably due to the fact that orthostatism favors venous engorgement in the sites corresponding to the territory of the inferior cava. In the femoral vein the normal average figure is 111 mm H.0.

Venous hypertension in the territory of the inferior cava is the cause of most of the clinical manifestations of right heart failure, of which the most typical is edema.

generalized venous hypertension

Generalized hypertension is observed in the following cases:

1. Muscular effort. It can produce appreciable increases in venous pressure (200 mm H,0 or more). It is a temporary increase that slowly decreases.
2. Right heart failure. The inability of the right heart to send all the blood it receives from the venous system to the pulmonary tree determines an increase in systemic venous pressure (150-250 mm H,0).

3. Pericardial processes. Pericardial symphysis or large effusions, which cause an impediment in diastole (hypodiastole).
4. Mediastinal alterations. Exudative or productive mediastinitis, also called fibromediastinitis.

5. Rheumatic or syphilitic heart disease.
6. Pulmonary scleroemphysema with chronic bronchitis. Even when it is not accompanied by chronic cor pulmonale, that is, impact on the right chambers (heart failure). These cases frequently present mediastinal fibrosis.

7. Tricuspid insufficiency.

CARDIAC IGATE. REFLU 0 EPATOJUGULAR

Hepatic congestion is only the accumulation of blood in the liver, especially in the left lobe, because its veins flow into the cava more perpendicularly and closer to the heart. This passive congestion is manifested by increased sensitivity of the gland, especially in the epigastrium as it corresponds to the left lobe, and an increase in size that varies with the degree of stasis (accordion liver).

Compression with the hand determines the exit of a certain amount of blood into the venous system, and the jugular veins, generally already engorged in these circumstances, become even more engorged (hepatojugular reflux).

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PERIPHERAL VASCULAR SYSTEM. ANATOMY AND CLINICAL PHYSIOLOGY. ANAMNESIC DATA AND MAIN SYMPTOMS

CLINICAL ANATOMY AND PHYSIOLOGY

The anatomy of the heart, arteries, veins, and lymphatic vessels has been described in Chapter 10.

The fundamental mission of the vascular system is to fulfill the circulatory function of supplying oxygen and providing energy to the tissues, eliminating excretion products, distributing the elements secreted by the endocrine glands and regulating body temperature. To do this, it has a driving system formed by the heart and arteries, a collection and return system integrated by the venous system, and an extensive capillary network, capable of constantly undergoing modifications in its functional level to achieve exchange in the various organs and tissues.

Functionally, the vascular system is divided into four sectors:

1. Pressure reserve sector. Consisting of the aorta and vessels of medium caliber and larger, with a wall rich in muscle fibers, capable of retaining the energy transmitted by the heartbeat and achieving a better distribution of the pulsatile flow so that it reaches the tissues uniformly.

2. Resistance sector. Composed of small arteries, arterioles and arteriovenous capillaries. Peripheral resistance is basically regulated by the latter, capable of modifying the percentage of its rich network that is in operation, according to the irrigation needs of the organ or tissue at any given time. Together with the pressure sector it contains approximately 20% of the blood volume.

3. Exchange sector. Composed of the capillary bed, through which cellular exchange occurs. It contains 5% of the blood volume.

4. Volume reserve sector. Formed by the venous system, an authentic capacitance system that stores 75% of the circulating volume.

The regulation of the vascular system is exerted by nervous (sympathetic) control over the arterioles. However, small arterioles respond mainly to local biochemical changes, especially to nitric oxide, released in response to endothelial aggression caused by

blood flow changes. Other factors, such as prostacyclins, also act on the arterioles, although in a more delayed manner.

Here we will discuss the main functions of the arterial circulation of the extremities, which are:

1. Transport the substances that take part in normal tissue metabolism, with the aim of maintaining a normal state of nutrition in the most distal portions. From an anatomical point of view, the extremities are made up of skin, subcutaneous cellular tissue, muscles, nerves, tendons, etc. These structures, including muscle at rest, need very little amounts of elements to maintain their normal metabolism and nutrition. The exercising muscle, on the other hand, needs a great metabolic exchange. For a decrease in the circulatory flow of an extremity to affect the trophism of these structures, it is necessary that around 65% of the blood flow of said extremity has been reduced. Your symptoms will be:

   fatigue and intermittent claudication. Beyond this reduction in flow (65%) we will then find trophic disorders at the level of the skin, nails, muscles, and others.

2. Carry the immunoleukocyte defense elements that usually act against inflammation. It is for this reason that insufficient arterial circulation can be expressed, in practice, by a delay in the healing of a wound.

3. Regulate local and body temperature in general. Here the sympathetic system and vasoactive substances fundamentally intervene.
4. Regulate the relationship between content (blood volume) and continent (arterial and venous tube systems). Blood vessels are not rigid tubes, but have a certain degree of contraction of their walls known as vascular tone. Depending on the vasomotor state, the vessels may undergo a process of extreme vasodilation (vasoplegia), a process of moderate vasodilation (vasodilation) or vasospasm (vasOconstriction). This vasomotor tone is regulated by the autonomic nervous system and especially by the sympathetic system, the humoral systems and the parietal muscular system.

ANAMN PHYSICAL DATA

We will point out here some elements of the anamnesis that are of interest in the semiology of this system: sex, diet and lifestyle habits, toxic habits, work and occupation, and personal and family history.

SEX

It is important, because while disorders related to atherosclerosis are more common in men, varicose veins and Raynaud's disease are more common in women.

FOOD AND BITES OF LIFE

Here we must investigate, above all, if the patient maintains a high-calorie and high-fat diet, if he leads a sedentary life or if, on the contrary, he develops great muscular activity; In the latter case, you must know the type of physical exercises you perform, etc.

TOXIC BITES

It is known that the harmful effect of smoking causes obvious disorders in the distribution of blood in the tissues. Ergotism has the same effect in those subjects who abuse ergot derivatives (ergotamine), which can cause serious circulatory disorders in the lower limbs and lead to gangrene in the toes or another region.

Currently, in our country it is rare because it is not customary, as in other countries, to regularly eat rye bread in the diet.

WORK AND EMPLOYMENT

They must be investigated, since certain professions or jobs can modify the normal dynamics of irrigation or return.

PERSONAL BACKGROUND

Among them, diabetes is of great value, as this condition causes chronic degenerative vascular alterations; recurrent infections and lymphangitis or mycoses of the feet, which so easily predispose to a secondary infection; the presence of atherosclerosis in other regions: thrombophlebitis, angina pectoris and intermittent claudication, among others.

FAMILY HISTORY Family history of

cardiovascular diseases

It includes ischemic heart disease, cerebral vascular insufficiency, especially at an early age, arterial hypertension, peripheral ischemia with a history of intermittent claudication or gangrene. It is also important to establish if there are family members with metabolic risk factors such as diabetes, dyslipidemia and hyperuricemia.

The presence of varicose veins and edema of lymphatic etiology is common in more than one member of the family group.

After the anamnesis, take into account in the physical examination:

Inspection: the patient's vascular examination must be complete and never limited to the area where he reports his discomfort. It is necessary to assess the color of the skin (redness, cyanosis, paleness), the degree of hydration, the presence of varicose veins, edema, reddish (inflammatory) or ocher skin plaques (stasis dermatitis), ulcerated or necrotic skin lesions. Some functional tests that assess changes in the relationship with postural variations (Ratschow test) can be useful in the course of a clinical examination even though the proliferation of non-invasive and widely disseminated complementary examinations has made them fall into disuse.

Palpation: Palpation of the carotid, axillary, humeral, radial, ulnar, femoral, popliteal and distal arteries (pedial and posterior tibial arteries), as well as abdominal palpation in search of possible aneurysmal dilations, allows us to establish the permeability of the vascular tracts and rule out the existence of dilations or thrills in them. Skin temperature should be determined with the back of the hand; There are areas of coldness due to ischemia or increased temperature due to superficial phlebitis, lymphangitis, hypodermitis and other inflammatory processes. Palpation of the jugular vein of the superficial tracts of the upper extremities and, especially, the lower extremities, allows us to appreciate the degree of turgor of the venous network and identify thrombosed tracts that appear indurated and hot.

Percussion: it is a maneuver that is not very current in vascular exploration.
Auscultation: should begin over the cardiac area and continue in the supra-aortic trunks of the laterocervical and supraclavicular location, the abdomen and the inguinal regions.

MAIN SYMPTOMS

We will study the most frequent symptoms in these pathologies, such as: fatigue, cramps, pain, sensation of coldness, paleness, numbness and tingling, and burning sensations.

FATIGABILITY

It may be the beginning symptom of vascular disease. It often goes unnoticed if it is not investigated directly. It is of no value when it occurs as part of a general fatigue picture: but when it appears in the legs, with a persistent nature and not associated with great exercise efforts, it requires a more in-depth investigation of the vascular system. For what

It generally appears many months before other more prominent symptoms, especially in the case of occlusive arterial diseases.

CRAMPS

Muscle cramp or claudication is one of the most prominent symptoms, often the dominant symptom. It can be of two types: exercise cramp or intermittent claudication and rest cramp.

Stress cramp or intermittent claudication Concept

It can be defined as muscle pain with the character of spasmodic, involuntary contraction, which occurs during exercise and disappears with rest. It is a very important and often pathognomonic symptom.

semiography

It intensifies when walking quickly or uphill. Patients describe it as a feeling of tightness, tightness or cramping of the muscle masses of the calf.

Intermittent claudication can be mild and moderate at first, but if the patient persists in walking it can become so severe that it is necessary to stop walking to rest or massage the limb before being able to continue. Although the most common location is in the calf muscles, cramp-type pain can occur in the foot; In these cases the doctor must be prepared to make the differential diagnosis with metatarsalgia or other orthopedic disorders. It may also be located at the level of the hip (hip claudication), as is usually observed in very high arterial occlusions, at the aortic or iliac level. Intermittent claudication of an extremity may not manifest if for any reason the patient does not travel the distance required for the pain or cramp to occur. For the same reasons, exertional angina pectoris can hide intermittent claudication or vice versa.

Semiogenesis or pathophysiology

In general, it is accepted that pain in claudication is fundamentally due to a decrease in muscle circulation.

Lewis verified the effect of muscular activity under conditions of ischemia, by demonstrating that pain is not necessarily due to tonic contractions of the muscles, but rather the result of the accumulation of disposable products of muscle metabolism, as these act on the sensitive nerve endings at the level of the muscle masses, as well as that pain does not co-exist.

It does not begin as the result of circulatory arrest, but when the muscles have performed some work under conditions of circulatory insufficiency. Furthermore, the pain disappears quickly when the metabolites that produce it and that have been accumulated in the muscle are dispersed, once the supply of oxygenated blood to these muscular elements has returned.

Neal reported some interesting observations using arteriography immediately after exercise cramps occurred. He confirmed that pain and intermittent claudication are not due to arterial spasm and, to a certain extent, confirmed Lewis' ideas. According to him, the real explanation would be that there is a metabolic disorder or biochemical changes in the muscles' own nutrition.

Semidiagnosis

It is observed in early atherosclerosis obliterans and in Buerger's thromboangiitis obliterans.

Rest cramp

Concept and semiography

Claudication that occurs at rest is a fairly common symptom. Patients report that they are woken up at night by a cramp-like pain in the leg, which is relieved when they get out of bed, walk, or massage the affected part. It is very interesting to highlight how frequently the patients themselves forget this symptom, to such an extent that they do not usually express it voluntarily except when they are asked a direct question in this regard.

Semiogenesis or pathophysiology and semidiagnosis

Rest cramp is a particularly noticeable symptom in patients with atherosclerosis obliterans. Furthermore, nocturnal cramp can occur in severe diabetes, especially of the juvenile type with marked alteration of carbohydrate tolerance and where patients excrete large amounts of glucose in the urine. It is then known as the Unschuld sign. It is possible that this phenomenon is the result of muscle irritability due to decreased muscle glycogen.

This disorder can occur several times a night in cases of severe diabetes and disappears immediately after the administration of a diet rich in carbohydrates together with insulin.

Patients with varicose veins and stasis edema, and pregnant women, especially in the last few months, may suffer from nocturnal cramps; These may also be present in patients suffering from nicotinic acid deficiency, such as in pellagra, and in patients with hypoparathyroidism, in the latter case, as a consequence of the hypocalcemia characteristic of the disease.

PAIN

In peripheral circulation disorders, pain may be linked to:

1. Venous disorders.
2. Arterial disorders.
3. Nervous disorders (ischemic neuritis).

This pain must be differentiated from other neuritis, arthritis and mechanical orthopedic conditions.

semiography

Pain in the extremities in vascular disorders can vary in intensity from tolerable to acute, “excruciating” (tearing) pain. It is usually intensified by exercise, but there are conditions in which the pain can be intense, even during rest.

Semiognesis and semidiagnosis

It is not a simple matter to try to explain the mechanism of pain. We know that it is associated with vascular disorders and, however, it may be insignificant in some patients who have advanced pathological changes in the walls of their vessels. On the contrary, patients with small ulcers, frequently seen in atherosclerosis, can be observed who have acute pain. On the other hand, cases with very extensive gangrene can be observed where the pain is tolerable or the patient may suffer only slightly; This is especially seen in cases of diabetic gangrene. In syringomyelia, patients may have extensive trophic-type ulcers with a significant lack of pain.

In thromboangiitis obliterans, pain is a very important finding; It is almost invariably present and is often an early symptom in this group of patients; We probably have a combination of influences to produce the symptom, so that there are not only definitive changes in the arteries and veins, but the nerves that accompany the vascular structures are also affected.

Priestly demonstrated the presence of Walerian degeneration in the nerves of these patients and showed that these changes were due to interference with the blood supply to these nervous structures. Although peripheral neuritis is a frequent complication of diabetes, probably associated with a deficiency of vitamin B, there is a form of neuritis specifically related to atherosclerosis obliterans and which usually occurs occasionally in an extremity affected by circulatory insufficiency.

Cottrell has demonstrated the existence of thrombosis of the vasa nervorum in peripheral nerves. These lesions would disturb the nutrition of these structures, resulting in the fragmentation of the myelin sheaths and even the degeneration of the axons, both lesions being irreversible.

Ischemic neuritis is usually unilateral, it is present in that extremity affected by acute arterial obstruction or where there are manifestations of advanced atherosclerosis. It is characterized by continuous, severe, intractable pain at rest, which usually worsens at night. It is generally associated with paresthesias such as numbness. The examination is usually accompanied by osteotendinous areflexia, loss of sensitivity to the tuning fork and position sense, as well as hypoesthesia and even anesthesia.

The pain in thrombophlebitis can be intense and in some cases unbearable. The reason for the intensity of the symptom is not entirely apparent. The case of phlebitis associated with a large vasospasm of the accompanying artery is known, but this is not a completely satisfactory explanation. In embolic obstructions pain is almost invariably present. The degree and intensity will depend on the caliber of the occluded vessel. In small branches the pain may be slight and temporary. In older people it can be acute and prolonged. It is often the first sign. However, in some cases numbness and tingling in the pads of the fingers may appear a few hours before more important symptoms develop. When the major vessels are suddenly occluded, the pain is attributed to an extensive spasm that develops in the occluded artery and the vessels in its immediate vicinity. The sudden blockage of circulation gives rise to anoxemia in the tissues and muscles that are supplied by the occluded vessel and this is almost certainly the explanation for the pain in these cases.

Pain can be an annoying accompaniment in some cases of arteriospasm; It can also occur particularly in patients who have had severe injuries to the lower extremities. Subsequently the lesions may heal, there is apparent recovery, but the patient may complain of cold feet and discomfort. The examination reveals coldness in the feet, presence or absence of pedal pulses, tests of circulatory function with evidence of arteriospasm, but there is no occlusive vascular disease. Radiological examination demonstrates osteoporosis. Such is the case of the so-called reflex sympathetic dystrophies (Sudeck's atrophy).

Continuous and severe pain at rest in a finger or foot that is not improved by exercise or change in position is usually related to an infection in an ischemic limb. In these cases there would not be adequate blood supply to cause the other symptoms. us from inflammation: redness, heat and tumor. Severe foot pain that awakens the patient after 162 hours of sleep and that improves when walking or leaving the limb hanging from the bed is characteristic of severe ischemia. Sleep and lying position decrease blood pressure and cardiac output with consequent damage to circulation in distal areas.

FEELING OF COLDNESS

semiography

Patients often complain of coldness, particularly in the lower extremities. This is a common symptom, more evident during the winter months and occurs in both legs. When the coldness is unilateral or limited to a finger or some local area, this symptom has greater significance and a more in-depth investigation should then be carried out.

Coldness in the feet per se does not necessarily mean that there is a vascular disease, since normally the temperature of the feet is lower than that of other parts of the body. The doctor must be prepared to distinguish between the subjective sensation of coldness and the true drop in skin temperature, which can be verified using special thermometers, or when there are obvious (significant) regional differences; For example, between foot and leg on one side and the other. This symptom, due to its variety and the variations in skin temperature, should not be given much value when it occurs alone, except in those cases in which it appears in a very limited area.

Semiognesis and semidiagnosis

A large number of factors intervene in maintaining the level of surface or skin temperature under resting conditions. Ordinarily it is the result of the heat transmitted to the skin by the blood and the loss to the environment produced by irradiation or vaporization. Taking into account that the amount of heat transmitted to the skin depends largely on the speed of local blood flow, the reading of the surface or skin temperature can be taken into account as a qualitative index of the skin circulation through the part considered, as long as the environmental temperature remains constant.

Skin temperature readings that are lower than room temperatures suggest an increase in sympathetic activity capable of producing excessive sweating, as well as a reduction in skin circulation due to large local vasoconstriction. Readings that are 5° or 6° above the temperature

ture of the room, indicate a normal state of vasomotor tone. Others that are considerably higher imply increased cutaneous blood flow that is due to low sympathetic activity.

In the presence of an arterial occlusive disorder, there may be a low skin temperature. It is often necessary to determine the role of vasospasm in this response before attributing it to a change entirely caused by permanent structural damage to the blood vessels.
It is interesting to note that, sometimes, there is an apparent paradox, given by a higher skin temperature on an extremity with an organic arterial lesion than on the opposite side where the arteries are normal or less damaged. This is likely due to the production of a large efficient collateral circulation. Another important point to highlight is the fact that stenosis and even occlusions of the arterial tree can exist without a parallel reduction in skin temperature.

PALE

Concept and semiography

Solitary pallor is a common symptom. When it is associated with coldness and drowsiness, the patient becomes alarmed. However, the paleness of a finger can be a striking phenomenon in those patients who, due to their occupation, use it more, in workers who use, for example, the vibrating pneumatic hammer.

Semiognesis and semidiagnosis

Paleness of the fingers followed by cyanosis and then redness is characteristic of Raynaud's phenomenon. These episodes are reproduced both by exposure to cold and by emotions.

The sudden paleness of an extremity with pain and coldness is a common accompanying phenomenon of embolic occlusions. It can also be seen in chronic occlusive arterial disorders, such as thromboangiitis obliterans, atherosclerosis, and diabetic atheromatosis, but in these cases the pallor is associated with other characteristic symptoms that suggest a peripheral vascular disorder.

Elevation of the limb and, at the same time, exercises on the most distal parts of it can give rise to a paleness that would not have been apparent otherwise.
This test for plantar ischemia is important and usually implies impaired arterial circulation.

Pallor of the extremities may also occur in patients with vasospastic disorders. These patients may have cold feet, and although they are not necessarily cases of Raynaud's syndromes, many are classified as such. However, they do not present the characteristic cycle of color changes.

tion described and do not develop the complications of this disease.

NIGHTNESS AND TINLING

Numbness and tingling are included as symptoms in the peripheral vascular system because they are related to blood supply.

Semiognesis or physiopathology

Lewis made observations regarding the development of the numbness through compression of the large arteries and concluded that this symptom is a consequence of the interference of the blood supply to the nerve trunks and that the tingling in the foot pads is not necessarily due to the return of blood to the fingers, but rather to the recovery of ischenmia in the nerve trunks of the extremity.

Semiography and semidiagnosis

The upper extremities are more frequently affected and symptoms are usually bilateral.

When numbness and tingling are associated with other symptoms and signs, they can be attributed to secondary vascular disorders, such as:

1. Supernumerary cervical rib.
2. Anterior scalene syndrome.
3. Crutch arteritis, or the group of Occupational diseases.

These symptoms can also appear in the group of vasospastic diseases. De Takatz highlighted the importance of such symptoms in the onset of embolic occlusions. He reported that they appear several hours before the explosive symptoms of a large embolic occlusion manifest.
Unfortunately, it is not possible to make a conclusive diagnosis in every case of numbness and tingling. Circulatory function tests may reveal a variable degree of arteriospasm. The underlying condition is probably linked in some way to neuropathy.

UEMANT SENSATIONS

Concept and semiography

Sometimes the patient complains of burning pain in the extremities; However, it is rare for this symptom to be mentioned among the main ones. It is almost invariably referred to the lower extremities; It can be unilateral or bilateral.

Semiognesis or pathophysiology

It's not very precise. Some attribute it to hypersensitivity of the nerve endings in the foot.

Semidiagnosis

It is a characteristic symptom of erythromelalgia (Weir-Mitchell disease). In these cases, the burning sensations are very unpleasant and worsen when the limb is placed in a downward position or when heat is applied; On the contrary, the patient reports improvement when the feet are elevated or cold is applied.

Burning pains are not uncommon in patients suffering from thromboangiitis obliterans or atherosclerosis obliterans. In this group of cases, what is striking is the fact that despite the sensation of burning pain, the skin temperature is below normal, in contrast to the higher temperature observed in erythromelalgia.

ALTERATIONS OF THE PERIPHERAL VASCULAR SYSTEM IN THE PHYSICAL EXAM

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GENERAL ASPECT

The general appearance of patients may have little bearing on the diagnosis. Little information is obtained from examining the face other than the possibility of seeing a vascular tumor (angioma). The ears also usually offer little evidence, but we can occasionally find local gangrene in this region, particularly in Raynaud's disease. The oropharynx should be examined for evidence of inflammatory signs at that level. In some acute arteritis and especially in some phlebitis, the probable focus of origin has been located at this level.

Among the general symptoms, fever and tachycardia deserve particular attention. A fever of 39-40°C, usually preceded by a chill, usually accompanies acute lymphangitis. On the contrary, a light fever, true low-grade fever, usually between 37.5 and 38 °C, usually accompanies venous thromboses. This fact is so significant that it has been elevated to the category of a sign (Michaelis sign).

Tachycardia is a sign common to a series of peripheral circulation disorders. In venous thrombosis, its characteristic is that it is very high and is not related to temperature, that is, there is a dissociation between the febrile curve and the pulse rate.

This character gives it great diagnostic value in cases of venous thrombosis. This sign is known as the staircase pulse or Mahler's sign:

In cases of arteriovenous fistulas, the so-called Nicoladoni-Bramhan phenomenon may occur, which consists of a slowing of the pulse rate when the site of the fistula or the artery proximal to it is compressed.

INSPECTION

In both the lower and upper extremities, some aspects must be examined from the inspection point of view.

As:

-Color changes.

-Sweat changes.

-Trophic changes.

- Edema

- Injuries.

- Hemorrhagic extravasations.

- Epidermophytosis.

- Nodules.

-Collateral circulation.

COLOR CHANGES

Under physiological conditions, cutaneous venules and capillaries are almost entirely responsible for skin color. The greater transparency of the skin allows the color of the more deeply located subcutaneous veins to be revealed. The skin tint is fundamentally due to the relative amount of reduced and oxidized hemoglobin in the vessels of the study region, the number of capillary networks in the dermis and melanin. Blood enters the small vessels in an oxidized form and leaves them in a reduced form; therefore, the existing dye represents an intermediate state between both forms, largely dependent on the speed of blood flow.

Ruddiness

This color change is produced by a series of physiological and pathological states. Normally, a bright red arterial color indicates increased circulation and under this circumstance the skin is warm or hot.
Such a response is noted when the extremity is exposed to high environmental temperatures and the need for color dissipation arises, as reflected in arteriolar, capillary, venular and arteriovenous anastomosis vasodilation.

The flushing that occurs at the termination of a state of anoxia (reactive hyperemia) also represents a marked increase in cutaneous blood flow. The appearance of a transient flush when a limb is placed in a reclining position may be a normal or abnormal response; this change is due to relaxation of the subpapillary venous plexus followed by blood storage.

The differential point would be the fact that in the presence of decreased circulation there would also be a lower skin temperature. A cyanotic red color on a cold foot held horizontally is an abnormal sign. This may indicate some pathological process that causes the small amount of blood that reaches the fingers to be diverted to the venous side of the circulation without passing through the capillaries. Due to the high concentration of hemoglobin in this venous sector, there would be a tendency for the skin to assume an intense red color. Another possible explanation would be the existence of some abnormality in the enzymatic systems, which would prevent the release of oxygen from the blood as it passes through the tissues.

Cyanosis

This change in color also has various interpretations. A bluish color caused by exposure to a cold environment is generally a normal vasoconstriction response of the arterioles and veins.
This vasoconstriction causes stasis in the capillaries and venules, and facilitates the transfer of oxygen across the vascular barrier into the extracellular spaces. Invariably this change in color is accompanied by a decrease in skin temperature.

Cyanosis in the presence of a temperate environment generally indicates the existence of vasospasm in the arterial tree, particularly in the arterioles. The mechanism responsible is similar to that described in cases of a normal limb exposed to a cold environment, that is, a slowing of blood flow through the small vessels. An example of this type of response is that observed in acute deep thrombophlebitis, in disuse atrophies, in rheumatic pain disorders, in rheumatoid arthritis, etc.

The differentiation of cyanosis that has a vasospastic basis and that due to an organic disorder in the vessels rests on its disappearance; In the case of a vasospastic disorder, it disappears after the vasomotor influx is blocked through sympathetic anesthetic infiltration.

There are many other causes of cyanosis. The rapid appearance of cyanosis when the limb is placed in a downward position means a decrease in local arterial circulation; The change in color is due to the existence of large amounts of reduced hemoglobin in the blood that is retained in atonic superficial vessels. The presence of warm, cyanotic skin in contrast to the decreased skin temperature seen in vasospasm suggests that the responsible mechanism is some systemic abnormality, such as congenital heart disease or some difficulty in oxygenating the blood itself that is not taking place because of some pathological process in the lungs.

Pallor

We already said in the previous chapter that solitary pallor is a common symptom and that it is often associated with coldness and drowsiness. This type of change in a limb can have different implications depending on the type of abnormality to which it is associated. A normal or high skin temperature, associated with paleness, indicates that there is an adequate speed of blood flow through the cutaneous vessels, but that there is some constriction of these.

A similar response may be noted in the presence of a marked reduction in circulating hemoglobin,

as a result of some type of blood dyscrasia in which the speed of peripheral circulation is not affected or may be increased. A pale and cold extremity, however, means a definite reduction in cutaneous blood flow with emptying of the subpapillary venous plexus. Under such circumstances, the pallor may be intensified by elevation of the limb. This maneuver helps to drain venous blood from the extremity at the same time that the entry of arterial blood is more difficult because it flows against gravity.

SWEAT CHANGES

iperidrosis

Hyperhidrosis is determined by inspection and palpation. Sweat glands are innervated by sympathetic fibers. Therefore, hyperhidrosis (unless there is a large increase in environmental temperature) generally indicates sympathetic hyperactivity, which is why it is usually associated with vasospastic signs and symptoms, that is, coldness of the skin and cyanosis.

Additionally, hyperhidrosis and vasospasm may be seen in organic arterial disease, such as thromboangitis obliterans.

An hydrosis

The loss of sweating can be produced by different mechanisms. They are: injury to the sweat glands themselves; dermal atrophies; elimination of sympathetic control, by sympathectomy or by complete destruction of a peripheral nerve. If the blood supply is reduced due to organic arterial injury, the sweat glands affected by anemia will reduce their secretion. In contrast to the cold and wet extremity, which indicates vasospasm, we would have the cold and dry extremity that is associated with decreased blood flow due to occlusive arterial disease.

TROPHIC CHANGES

Trophic changes in the skin cannot be noticed given its poor appearance. Tightness in scleroderma is easily recognized.

Nails and hairs

Nails should also be inspected for trophic changes, given the great influence of circulation on nail growth. The most frequently observed alterations are: delay in growth, loss of brilliance and changes in color.

Delayed hair growth or hair loss is also usually an expression of decreased blood flow.

EDEMA

It is usually an accompaniment to various vascular disorders. It can appear under physiological conditions, such as postural edema, edema linked to large increases in environmental temperature and that observed in obese people. On the other hand, edema may be related to some pathological conditions such as thrombophlebitis, occlusive arterial diseases, vasospastic states and local inflammation. In addition, there are systemic disorders that can lead to the production of edema in the extremities, such as congestive heart failure, hypoproteinemia, and sodium retention.

Semiognesis or pathophysiology

In thrombophlebitis, the mechanism responsible for the formation of edema is interference with blood return, produced by occlusion of the main venous trunks. As a consequence, there will be stasis in the small veins and capillaries followed by an increase in their pressure, which sometimes reaches a figure five times higher than normal. This increase in pressure determines the increase in capillary filtration pressure, which gives rise to the passage of fluid from the bloodstream to the extravascular spaces. As a consequence of the venous stasis itself, there may be some degree of anoxia of the capillary wall, producing increased permeability, which in turn contributes to greater fluid transudation.

There is also interference with the lymphatic drainage of the extremity which is responsible for some of the edema in acute thrombophlebitis. This happens, in part, due to inflammation of the neighboring lymphatic ducts caused by phlebitis and periphlebitis.

Another factor that participates in the production of lymphatic stasis is the spasm of medium and small arteries, produced by a vasomotor reflex, which begins at the level of the segment of thrombosed veins, and which acts as an irritative focus. As a result, there will be a reduction in the amplitude of the pulsations of the arterial tree on the neighboring lymphatics, which are normally filled and emptied as a consequence of the pumping produced by the distention and rhythmic narrowing of the arteries in each cardiac cycle.

The vasospasm that accompanies acute deep thrombophlebitis adds another mechanism to this edema. In addition to the effect on lymphatic drainage, arterial spasm produces anoxia of the capillary endothelium that causes increased permeability, and, consequently, increased fluid transudation, since it favors the movement of fluid from the vascular bed towards the perivascular spaces and at the same time prevents

changes that occur or tend to occur in opposite directions. The edema that appears as part of the postphlebitic syndrome, when the patient becomes ambulatory again, reflects an insufficient venous system, unable to cover the return of blood from the lower extremity when the patient assumes the erect position.

Elimination of vasomotor tone by temporary or permanent sympathectomy generally results in a reduction or elimination of edema.

INJURIES

In cases of arterial insufficiency, various injuries can be found on the limbs. They are:

pink spots, scars, blisters, ulcerations and areas of gangrene.

Pink mancs

They are small, pinkish pigmented areas, usually seen on the legs or the back of the feet.

They are small and few in number.

Scars

It is not uncommon to find scars on the leg in patients suffering from arterial diseases. The scars are usually clear. Some are small depressions that are seen under the skin, discreet, slightly spaced, but in many cases, numerous. They are common in diabetics with poor peripheral circulation. Some of these scars may be pigmented.

Flictenas

The presence of flictenas is extremely significant.

They have a size ranging from 1-5 cm. The small ones are rounded, unlike the large ones, which are oval; It is more common to find them near the fingers. However, they can also appear on the back of the foot. A flictena can be due to burns, either by immersion in hot water or by exposure to electric lamps with the aim of producing an increase in heat in the extremity. Flictenas found in patients with arterial diseases have certain characteristics: they almost always appear at night or develop very quickly.

The patient generally cannot explain his development. They are usually asymptomatic, except when they give a certain burning sensation. They do not show signs of an inflammatory reaction and, as a rule, when they are discovered they have already reached their complete development; they do not extend.

The blisters that appear in diabetics also have their characteristics: they are white or colorless; Usually within 48 hours they become purpuric; Shortly before that, its bases become gangrenous. Large blisters are also observed in patients who rapidly develop massive gangrene due to other causes.

and particularly in embolic occlusion of the great arteries.

ulcers

They generally occur in those areas that receive constant trauma or irritation from shoes.

This occurs at the base of the first stem or at the base of the smaller stems. These lesions are common in atherosclerosis. Small ulcerations can be found between the fingers. A history of recurrent ulcerations should raise suspicion of poor circulation. When these ulcerations are larger and symmetrical, syringomyeli should be suspected.

Gangrene

This term is used to indicate mass tissue death and, therefore, represents an intense trophic disorder; it is not a disease but only a symptom common to many diseases. It comes in the following forms: focal, dry and wet.

Focal gangrene

It generally covers a small area and is frequently located on the fingers, near the nails. Sometimes it takes the front face of the first artery, so it could escape the patient's vision.

These small, localized areas of focal gangrene have no tendency to spread. Its presence means the existence of pathological changes in the small vessels. It is more common in diabetics, although we can also find it in attacks of thromboangiitis and in atherosclerosis obliterans in an advanced stage (IV).

dry gangrene

It is one where the necrosis is in the tissue mummification phase and appears as a dry, black scab, firmly adhered to the deep planes. It is almost always limited by a clearly marked line and the tissue surrounding it has a normal color, with no signs of infection. This form of gangrene may be the result of an acute attack of ischemia or the last stage of acute infectious gangrene, in which the infection has completely stopped. It could also be the last stage of any form of traumatic gangrene in which the infection has been controlled.

As a prominent feature, this form of gangrene appears hard, black and dry. When vascularization is poor, gangrene will persist for several months. The neighboring, relatively vascularized tissues create a spontaneous separation of the affected tissue, through liquefaction of the necrosis at the margin of the lesion. Treatment of the disorder depends on the recognition of

this biological characteristic, due to which spontaneous amputation occurs.

Wet gangrene

This is the form of gangrene that predominates in diabetics and thromboangiitis. Wet gangrene is not only characterized by infection of necrotic tissue, but in it we always find a certain degree of infection of the adjacent tissue, which when sectioned, will show fat necrosis in the subcutaneous cellular tissue, in the form of a malodorous exudate that contains fat globules and some gas bubbles. We will also find some important tendons or necrotic bones. Bleeding will be light or absent. If we explore more deeply we will find viable tissue, which is edematous and from which a fine serosanguineous exudate flows. This is precisely where the essence of the problem lies since it is in this viable tissue where the pathogenic bacteria develop.

The success of the treatment is based on the destruction of the organisms that maintain this continuous state of latent or latent cellulite.

Examining the lesion we observed a necrotic eschar with a weak line of demarcation and a large area of tissues that reveals evidence of active inflammation. The skin overlying the viable area varies from erythematous to violaceous, as well as edematous and soft. The necrotic eschar is thin, discolored and foul-smelling; In it it frequently forms a sinuous path from which a purulent exudate will drain. The patient does not present symptoms of poisoning, and may continue for a period of weeks or months with a relative degree of color and slowly progressing gangrene.

The proportion of the degree of destruction in these cases depends on several factors: first, the virulence of the organism producing the infection, and second, the degree of circulatory insufficiency.

Chronic wet gangrene transforms into dry gangrene when all traces of infection disappear.

EMORRICAL EXTRAVASATIONS

Hemorrhagic purpura is sometimes seen in the upper and lower extremities of patients with acute arteritis. Likewise, they can be observed in some cases of periarteritis nodosa.

EPIDERMOPHITOSIS

Skin lesions with fissures between the toes and itching may indicate the presence of epidermophytosis. This is of particular importance not only as a gateway for pathogenic bacteria in triggering the acute lymphangitis crisis.

da, but also because they are usually the starting site of focal gangrene lesions.

NODULES

They can be seen on the extremities particularly in patients with periarteritis nodosa. Other times, they have a frank inflammatory basis as observed in the so-called nodular vasculitis, generally produced by toxoallergic mechanisms. Sometimes they ulcerate, as in erythema induratum of Bazin.

COLLATERAL CIRCULATION

The presence of a superficial collateral venous circulation is often a revealing sign of the existence of deep thrombophlebitis. It is more frequently observed at the level of the pretibial region (Pratt's sentinel veins), in cases of thrombophlebitis of the deep veins of the leg, or at the root of the thigh in cases of iliofemoral thrombophlebitis.

PALPATION

By this method the examiner not only verifies what has been reported in the patient's medical history and inspection data, but also adds some information that can help in the diagnosis. To do this, you must verify the presence of:

-Trophic changes.

-Nodules.

- Phlebitis.

- Coldness.

- Heat.

The examiner must also perform the examination of arterial pulses and nodes.

TROPHIC CHANGES

When we palpate the distal parts, trophic changes of the skin can be noted. The tightness of the skin in scleroderma and the thinning of the underlying cellular tissue cutaneous are easily recognized.

NODULES

They are usually palpated in the extremities, located along the course of the arteries. They are small and almost always very painful. Nodules are of great importance when the possibility of periarteritis nodosa is raised. However, the diagnosis cannot be definitive until its histological signs are recognized after a biopsy.

Nodules can be detected in some patients with infectious recurrent thrombophlebitis and in other conditions associated with vascular disorders such as erythema induratum, nodular vasculitis and erythema nodosum.

PHLEBITIS

It is clearly evident when the superficial veins are affected. In many cases the deep veins are involved, and the inflammatory reaction does not reach the surface. In some cases of superficial phlebitis we can palpate the venous thrombosis as a line of induration of the skin. Recurrent and migratory superficial phlebitis is present in approximately 40% of cases of thromboangiitis obliterans.

COLDNESS

The coldness of the skin is confirmed by palpation. The examiner's hands should be warm. The dorsal aspect of the second phalanx is preferred for this examination. Many believe that this symptom can be satisfactorily explored by palpation, without the need to use special instruments, although for greater accuracy and precision, instruments such as the electric thermometer have been built that allow not only the exact obtaining of the skin temperature in a given location, but also obtaining the temperature differences between this and any other chosen location.

When coldness is symmetrical and bilateral, it is due to a decrease in circulatory flow, either due to a vasospastic condition or an occlusive vascular disorder. When only one finger is cold, it may be due to a marked degree of persistent vasospasm or an organic condition of the arteries. When it comes to local disseminated areas, these may be caused by a neurological condition.

HEAT

Often a patient may complain of burning sensations, but does not necessarily show an increase in temperature of the feet or a foot. When the burning sensation is associated with occlusive vascular disease, the surface temperature is usually below normal. When heat is detected during an examination, cellulitis or phlebitis must be excluded. An increase in foot temperature, particularly when associated with marked redness, suggests erythromelalgia. The finding of an increased temperature in one extremity and not in the other indicates an increase in circulatory activity and in these cases the possibility of the existence of an arteriovenous fistula must be kept in mind.

SENSITIVITY

There may be some tenderness along the veins in cases of subacute phlebitis. We can find more or less sensitivity along the arteries in periarteritis nodosa.

TRILL

Thrill appears in patients with arteriovenous fistulas or aneurysms. It is more quickly recognized in the acquired form of arteriovenous fistulas secondary to gunshot or knife wounds.

EXAMINATION OF DENOPATHY NODES

The nodes in the inguinal region should be investigated. They can be helpful in some cases where the differential diagnosis must be made between thrombophlebitis and lymphangitis. In this last condition, satellite inguinal adenopathy is almost constant.

AUSCULTATION

It is another means of examination in peripheral vascular diseases, although of less value than the previous ones.
It is very useful in determining murmurs in patients with arteriovenous fistulas. In the case of hemihypertrophy of a lower extremity in a child in whom the diagnosis is obscure, the finding of a murmur in the groin can help confirm the suspicion of a congenital arteriovenous fistula. Auscultation should also be performed to verify the diagnosis of an aneurysm, much more so if it is located in the popliteal region.

The murmur in arteriovenous fistulas has the characteristic of being continuous with systolic reinforcement. In aneurysms it is a pure systolic murmur.

Auscultation of the aorta and the bilateral iliacofemoral sector allows us to detect stenotic lesions beginning in such arterial sectors, by listening to murmurs of different intensity that reveal the turbulence of the flow when passing through the arterial narrowings.

MEASUREMENT

The measurement of the circumference of the extremities provides certain information that can help in the diagnosis. In definite cases of thrombophlebitis, where the diagnosis is obvious, it is usually not so important, but occasionally when the diagnosis is not very clear, an increase in the measurement of a limb may induce suspicion of deep thrombophlebitis. In congenital arteriovenous fistulas, the affected limb has

a tendency to grow more rapidly; This growth includes the bony parts and the soft parts. The verification of said elongation therefore has great diagnostic value. The measurement of the circumference of the leg or thighs during the course of thrombophlebitis with marked edema can provide additional information regarding the evolution of the disease. In some patients the artery can be palpated some distance lower, even near Hunter's canal.

Normally the femoral pulse is full and strong. We should find it reduced in arteriospasm and abolished in all aortic occlusions or the corresponding iliac sector.

In dissecting aneurysm it may be reduced or absent.

ALTERATIONS OF ARTERIAL PULSES

The examination of peripheral pulses was discussed in Chapter 12 of Section I.

We will move on to study the alterations that we can find in the walls of the artery, as well as those in the arterial beat itself or arterial pulse.

ALTERATIONS OF THE ARTERIAL WALL

Semiography and semidiagnosis

The arteries, under normal conditions, offer no resistance to the finger that palpates them. In cases of arteriosclerosis, they are hardened and flexible, and can move as if they were rigid tubes, mainly the brachial arteries. On other occasions we find what is called chicken trachea artery, mainly in the radial artery, produced by atheromatous plaques, calcified or not, separated by membranous areas.

When we palpate the arterial pulses we must recognize evidence of sclerosis. This can be easily found if we examine the pedis or radial pulse, but not when it comes to deep vessels. Hardening of the femoral and brachial arteries can be revealed with careful examination. However, the presence of sclerosis should not be interpreted as synonymous with occlusive vascular disease, unless other symptoms and signs are detected. It is possible for an individual with sclerosis of these vessels to have efficient circulation and maintain satisfactory nutrition of those parts. Furthermore, sclerosis of one vessel does not necessarily indicate that others are also affected.

upper extremities

In the upper extremities the radial artery is the vessel usually examined and the axillary pulse is the one it is less frequently investigated. However, when there is suspicion of a vascular disorder of the upper extremities, because the radial pulse is absent due to an abnormality or circulatory insufficiency, the brachial artery and finally the axillary artery should be examined.

When the radial pulse is absent, it may be an anomaly, as long as there are no symptoms of circulatory insufficiency.

Among vascular disorders in which the radial pulse is not palpable, the following should be mentioned:

1. Vasospastic disorders.
2. Thromboangiitis obliterans.
3. Endarteritis obliterans.
4. Embolic occlusions.
5. Cervical rib.
6. Anterior scalene syndrome.

The absence of the humeral pulse may be due to:

1. High embolic occlusion.
2. Chronic arteritis.
3. Vasospastic disorders.

lower extremities

The pedis and posterior tibial pulses, with their clinical significance, were also described in Chapter 12 of Section I.

The importance of confirming the presence or absence of the popliteal pulse is evident when we are faced with a case of embolic occlusion and we need to specify the exact location of this.

We should find the femoral pulse decreased in arteriospasm and abolished in all aortic occlusions or the corresponding iliac sector. In dissecting aneurysm it may be reduced or absent.

SYNCHRONISM ALTERATIONS

The equality in the amplitude of the pulse of two arteries corresponding to symmetrical regions of the body can be modified, giving rise to what we call different pulses.

Right radial pulse delay

When we examine a patient and notice that the heartbeat of the radial artery in the right arm is delayed in relation to the heartbeat of the left arm, we must suspect the existence of an aneurysmal dilation of the brachiocephalic trunk.

Left radial pulse delay

When there is a delay of the left radial pulse in relation to the right, it may be due to the existence of

an aneurysmal dilation of the arch of the aorta, located between the right brachiocephalic trunk and the left subclavian artery.

Alterations in the synchronism of the femoral pulse with the radial pulse

The femoral pulse and the radial pulse are synchronous, therefore, when we find a delay in the femoral pulse in relation to the radial pulse, it may be caused by the existence of an aneurysm of the descending aorta or the abdominal aorta. On the contrary, when the femoral pulse advances in relation to the radial pulse, it is due to sclerosis of the descending aorta or the abdominal aorta.

ALTERATIONS OF THE PULSE WAVE CHARACTERISTICS

Frequency alterations

The physiological variations were already described in Chapter 12.

Tachycardia

It is the increase in pulse rate above the figures considered normal. Depending on the number of beats per minute we can distinguish:

Moderately accelerated pulse, when the frequency ranges between 90 and 100 beats/min.

Accelerated pulse, with a frequency between 100 and 130/min.

Very accelerated pulse, when there is more than 170/min.

Tachycardias are classified into two large groups: the sinus and the paroxysmal.

In sinus tachycardia the stimulus comes from the sinus node; It is characterized by its relatively moderate frequency, by beginning and ending gradually, by being proportional to the cause that originates it, and by decreasing with maneuvers or drugs that excite the vagus nerve. It can be observed in neurovegetative dystonia with sympathetic predominance; in fever; in poisoning by coffee, nicotine, alcohol or tea; in hyperthyroidism and in various decompensated cardiovascular diseases.

Paroxysmal or paroxysmal tachycardia is a rhythm of heterotopic, extrasystolic origin, which is characterized by the unusual frequency of the number of heartbeats, by its sudden beginning and ending, and by sometimes ending with manual maneuvers that stimulate the vagus (eye compression and compression of the carotid sinus).

Bradycardia

It is the decrease in pulse rate below the normal figures of 60 beats per minute.

It can be of different degrees:

Moderate, 50-60 beats/min.

Slow pulse, between 30-50/min.

Extreme bradycardia, with pulse less than 30/min.

True bradycardia can be of sinus origin and due to atrioventricular block.

Sinus bradycardia can be physiological in vagotonic individuals, who usually have great resistance to physical fatigue.

Pathological sinus bradycardia is seen: in cases of poisoning by barium salts, bromides, salicylic acid, etc.; in intracranial hypertension (it is a very important sign); in hepatocellular and obstructive jaundice and in hypothyroidism.

Bradycardia due to complete atrioventricular block is characterized by very low beats per minute, almost always less than 40, since the pacemaker in these cases is born in the ventricle (idioventricular rhythm).

Pulse rhythm alterations

Respiratory sinus arrhythmia

It was already described in Chapter 12 of Section I.

Intermittent pulse

If when we are examining a patient's pulse, we notice that a pulsation is missing and then the previous normal rhythm continues, we say that there is pulse intermittency.

This intermittency is generally due to isolated extrasystoles that fail to open the sigmoid valves. It is the so-called false intermittency. True intermittency is that in which systole does not occur due to there being a sinoatrial or atrioventricular block.

Extrasystolic pulse

When when examining the pulse we notice the appearance of a premature pulsation that is followed by a longer pause than the one between the previous pulsations, we have an extrasystolic pulse.

This pulse is produced by an extrasystole that opens the sigmoid valves, causing a pulsatile wave that appears before its time and is followed by a longer pause (compensatory pause) because, being the myocardium in the refractory period, it loses the next stimulus.

Complete arrhythmia

In these cases the pulse is completely irregular, the pulsations are different from each other and the intervals between them are also different; There is no rhythm, the inequality is complete.

In most cases, this arrhythmia is caused by atrial fibrillation, although it is not exclusive to this, as it can also be found in some cases of extrasystolic arrhythmias with disorderly grouped extrasystoles.

Fig amplitude alterations

Increased amplitude

This alteration practically corresponds to the increase in differential blood pressure. Aortic insufficiency is where it is best observed, since there is a phase of diastolic emptying, followed by a sudden and marked elevation during systole, which is why it has been called "jumping pulse" or celery pulse.

A wide pulse is also observed in hyperthyroidism and high blood pressure.

Decrease in amplitude

The pulse width is decreased in those cases in which there is a small differential pressure, most of the time accompanied by a decrease in blood pressure.

It is observed in cases of arterial hypotension and peripheral vascular collapse, in which the pulse becomes almost imperceptible (filiform pulse); It occurs in cases of shock and hemorrhages.

In patients with aortic stricture we also find a decrease in pulse amplitude.

Amplitude alternation

In some cases we observe that a strong and a weak pulsation alternate, but maintaining the same constant separation between one and the other, this phenomenon is called alternating pulse. It is caused by variations in the contractile force of the ventricles or their muscle fibers according to the myogenic theory, which is the most accepted; or due to variations in ventricular filling and aortic diastolic pressure.

Alternating pulse is an important sign of left ventricular claudication. It is generally a sign of a poor prognosis.

It must be differentiated from the bigeminated or bigeminal pulse, in which there is also a strong pulsation followed by a weak one, but the latter is separated from the first by a shorter interval than that which separates it from the strong pulsation that follows it and is produced by an extrasystole (bigeminated rhythm).

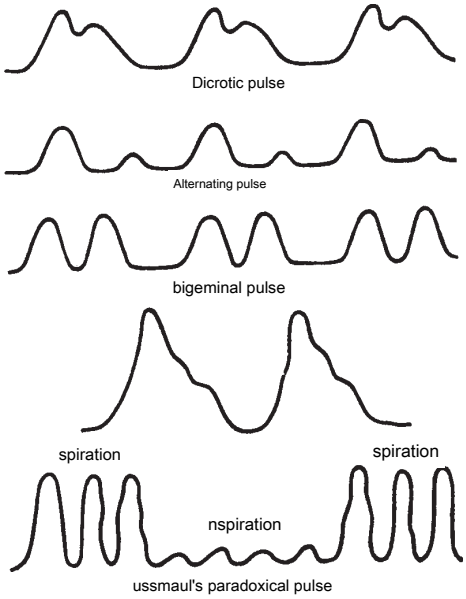


Fig. 47.1 Varieties of the arterial pulse.

Changes in pulse hardness

We already referred to them in Section I, but it does not hurt to reiterate them.

Hard pulse. When the emptying of the radial artery is difficult when compressing between the fingers. It is usually found in high blood pressure and arteriosclerosis.

Soft pulse. A soft pulse exists when when you compress the artery with your fingers, it depresses and empties easily. It is detected in cases of shock and after hemorrhages.

BLOOD TENSION ALTERATIONS

The semiogenesis or pathophysiology of blood pressure was discussed in Chapter 12 of Section I.

As a summary, we can say that blood pressure is maintained thanks to the heart and circulatory system, but at the same time there are external and internal factors, the latter governed by the vegetative nervous system;
Both influence blood pressure. Changes in these factors will cause this blood pressure to change in the direction of raising the blood pressure levels (arterial hypertension), or in the direction of decreasing them (arterial hypotension).

High blood pressure was studied in Chapter 45, as high blood pressure syndrome.

ARTERIAL IPOTENSION

Systolic and diastolic pressures are below the minimum limits, estimated at 100 and 60 mm Hg, according to Baunch, and 90 and 50, according to Hamilton.

Hypotension is classified as primary, secondary and orthostatic.

Primary hypotension is of unknown etiology and is found in 25-30% of the population. It is common in women and in asthenic subjects.

It can occur with minimal discomfort, or sometimes the so-called hypotonic symptom complex occurs, caused by headaches, vertigo, tinnitus, general laxity and difficulty concentrating, especially on morning tasks.

Secondary hypotension is observed in endocrine disorders (such as Addison's disease, Sheehano syndrome, Simmonds' disease), chronic pulmonary conditions (tuberculosis, emphysema), aortic stenosis, mitral stenosis, etc. (Fig. 47.2).

Orthostatic hypotension appears when the subject is sitting or standing, never lying down. It is a consequence of the stagnation of blood in the pending parts of the body, due to the failure of the compensation mechanisms, the disorders are more accentuated during the morning and attenuate during the course of the day.

When the reduction in systolic and diastolic volumes, as a result of orthostatism, reaches a certain limit, the so-called postural or orthostatic collapse occurs, with intense tachycardia and a state of pre-collapse or true collapse, which can lead to loss of consciousness.

ALTERATIONS OF THE PERIPHERAL VENOUS SYSTEM

Functional examination for the detection of alterations nes in the venous system in the lower limbs will be treated in Chapter 48 and the main venous syndromes in Chapter 49.

EXAMINATION OF THE NECK VEINS

Examination of the neck veins allows us to evaluate the characteristics of the jugular venous pulse, and the state of the central venous pressure (CVP), so that we can make judgments about the function of the right side of the heart.

Jugular venous pulses and CVP are explored by simple inspection or with invasive monitoring equipment capable of producing pressure waves. Simple inspection consists of observing the column of venous blood in the internal jugular vein. The pulsation of the internal jugular vein can be distinguished from the pulsation of the carotid vein by their differences in the type of beat.

The jugular venous pulse is characterized by several positive beats of small amplitude, as opposed to a vigorous beat of the arterial pulse.

To make this distinction, palpate the carotid artery on the opposite side while visualizing jugular venous pulsations. An alternative method is to ask the person to lie completely horizontal, which causes visible jugular venous distention. When the person sits down, the pulsations will disappear, because the veins collapse. Internal jugular venous pulses can also be identified by applying pressure to the neck, parallel to and just above the clavicle. Within 20 seconds, the vein will fill and the distention will become very evident.

Position and lighting

The person being examined should be observed from the right side, because the pulsations of the right internal jugular are more visible due to their proximity to the right heart. The person should lie down with the chest elevated between 30°-60°. The flat position is not appropriate, because if the veins are very distended the pulsations will not be able to be distinguished.

The head should be turned slightly to the left. Tangential lighting is recommended to accentuate shadows and make jugular venous pulses more visible.

Jugular pulse waves

It takes considerable skill to clearly see the waves by inspection, although it is relatively easy to detect them on a graph.

Three positive waves are distinguished: a, c and v.

The a wave reflects atrial systole; The c wave may be a distinguishable wave, or appear as a notch on the a wave, or be absent. The a wave is the largest positive wave and can increase in amplitude during inspiration.

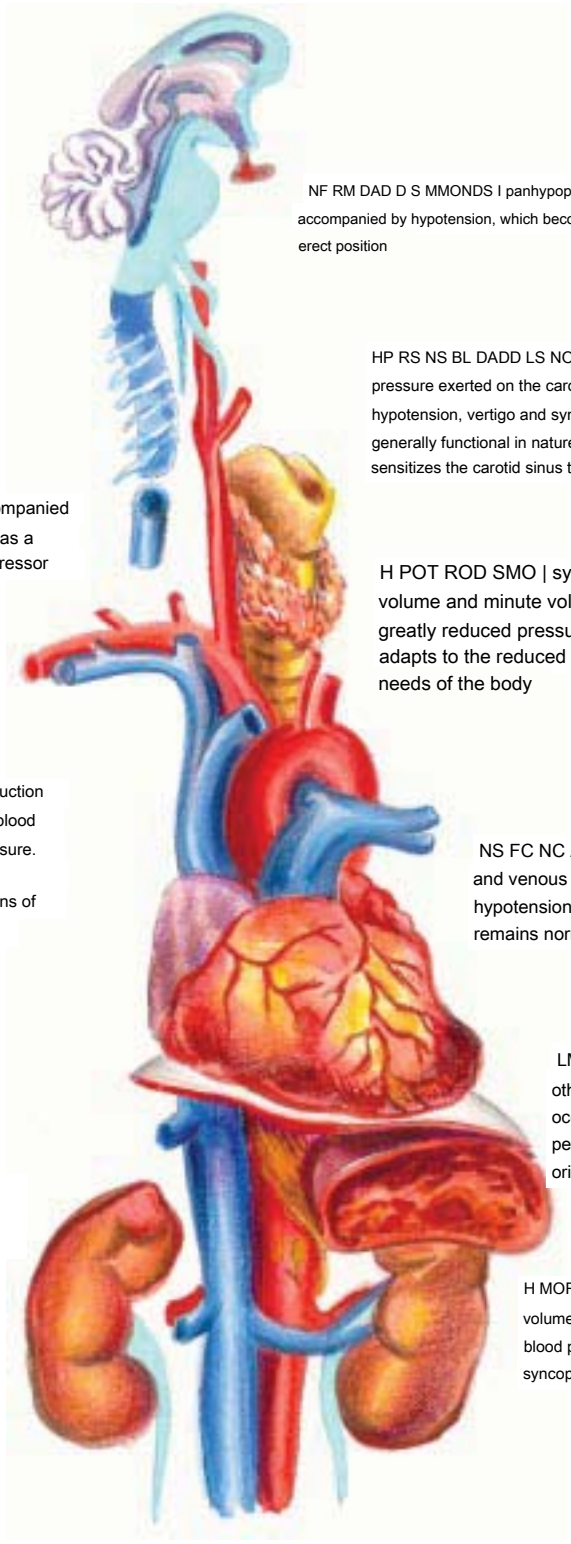
The ondac represents the closure of the tricuspid valve.

Wave v represents right atrial filling.

Negative waves include x and y. The decrease x occurs with ventricular systole, as the venous blood column declines. Y descent occurs when blood from the right atrium flows rapidly into the right ventricle.

Nornal jugular pulse. Some jugular venous pulse waves can be observed, reflecting normal pressure changes on the right side of the heart.

Abnormal jugular venous pulse. Conditions that increase resistance to ventricular filling, such as tricuspid stenosis, right ventricular failure, pulmonary hypertension, and pulmonary stenosis, can cause an increase in the amplitude of the a wave (a waves, cannon). Decreased amplitude of the a wave may be observed in atrial fibrillation and ventricular pacemakers. The



NF RM DAD D S MMONDS I panhypopituitarism is accompanied by hypotension, which becomes intense in the erect position

HP RS NS BL DADD LS NO CAROT D 0 The pressure exerted on the carotid sinus causes hypotension, vertigo and syncope. It is generally functional in nature. Digitalis sensitizes the carotid sinus to pressure.

SRNGOM LA May be accompanied by orthostatic hypotension as a result of paralysis of vasopressor reflexes

H POT ROD SMO | systolic volume and minute volume are greatly reduced pressure blood adapts to the reduced metabolic needs of the body

ST NOS SA RT CA The reduction in minute volume decreases blood pressure and differential pressure. Severe stenosis may be accompanied by manifestations of insufficient cerebral irrigation.

NS FC NC A CARD ACA Heart failure and venous congestion produce hypotension even when blood volume remains normal

LMOCARD O INFART Hypotension and other manifestations of 0C due to coronary occlusion may result from heart failure or peripheral circulatory collapse of reflex origin.

NF RM DAD D ADD SON Severe cortical adrenal insufficiency modifies the electrolyte balance, produces hemoconcentration and reduces blood

H MORRAG A The decrease in sanguineous volume reduces the minute volume of the heart and blood pressure, vertigo, profuse sweating and syncope may appear.

Fig. 47.2 Causes of secondary hypotension.

Tricuspid regurgitation can increase the amplitude of the v wave. Cardiac tamponade can cause an increase in both the a wave and the v wave.

Steps to follow for the examination of the neck veins

1. Observe the jugular venous pulsations by va-
several cardiac cycles.

Try to identify the a, c and v waves.

2. Estimate CVP by measuring the height of the pulsation in the internal jugular vein.

a) Choose a standard reference point from which to measure the height of the pulsation in the internal jugular vein.

The zero reference point, at the level of the right atrium, can be difficult to determine with certainty. Therefore, use the sternal angle, which is approximately 5 cm above the right atrium, as a reference point.

b) Measure the distance in centimeters, from the sternal angle to the height of the distended jugular vein.

c) Add 5 cm to the value obtained for a rough estimate of CVP.

The normal PVC is 3-5 cm above the sternal angle, when the trunk is elevated 30°-60°. Elevated CVP can be attributed to right or left heart failure, pulmonary hypertension, pulmonary embolism, or cardiac tamponade.

3. Look for hepatojugular reflux.

When the person rises 30°-60°, compress the right upper quadrant for 30-60 s with your palm.

Hepatojugular reflux is positive if the jugular venous pulse level rises with this maneuver.

Do this maneuver if you suspect right heart failure.

Increased abdominal pressure increases venous return to the right side of the heart. If the right ventricle is compromised, the pressure in the neck veins will increase.

COMPLEMENTARY EX MENES AND EXPLORATION

FUNCTIONALITY OF THE PERIPHERAL VASCULAR SYSTEM

48

Introduction

A provisional diagnosis of vascular diseases can be obtained from the history and a thorough physical examination including investigation of pulses and sclerosis of the peripheral arteries. However, neither a symptom nor the mere fact of examining the pulses is reliable, since we know that the main symptoms such as cramp, coldness and paleness do not exist in all patients with vascular disorders. It is also known that a patient can have some symptoms and yet have efficient circulation. A good pulse can be found, even in the presence of gangrene in diabetic patients, or be absent due to anomalous location of the vessel and not be evident in some small vessels even when collateral circulation in that part is efficient.

Hence the importance of studying complementary examinations and circulatory function tests. Among them the fundamental ones are:

1. Study of muscle circulation:

Claudication time.

Claudication distance.

Kisch test.

2. Study of capillaries and small arteries:

Plantar ischemia test.

Paleness-blush test (Pratt).

Angle of circulatory sufficiency.

Reactive hyperemia.

Capillary microscopy.

3. Study of the great arteries:

Oscillometry and oscillography.

Venous filling time.

Allen test.

Measurement of systolic pressure of the extremities.

4. Study of vasomotor tone:

Skin thermometry test.

5. Measurement of blood flow by non-invasive methods:

Doppler studies: pressure index, segmental pressures.

Digital plethysmography.

Alternating compression plethysmography.

6. Radiographic examinations of the vascular tree:

Simple plate of blood vessels.

Angiography (aortography, arteriography, phlebography and lymphography)

7. Study of the superficial venous system, the communicating vein system and the deep venous system:

Venous flowmetry for the study of the superficial venous system and the communicating system or venous-venous flowmetry (study of the deep venous system).

Double flowmetry (Duplex scanning) (for study of both systems).

Other tests:

Circulatory speed.

Radioactive isotopes.

STUDY OF MUSCLE CIRCULATION

To study muscle circulation, we have three tests:

- Claudication time.
- Claudication distance.
- Kisch test.

Claudication time

In general, this time takes into account the distance that the patient can walk comfortably before the painful cramp appears and the patient must rest. The patient says that he can walk 100 m, maybe 200 or 300; This information is very vague and it is necessary to obtain more exact information. Several procedures have been suggested, which not only indicate the degree of claudication, but can be used comparatively to assess the therapeutic effect.

The test is performed in the following way: after the patient has rested for half an hour, he is instructed to walk on a smooth floor at a rate of approximately 120 steps per minute. When any type of pain or cramp occurs, the patient is told to stop walking. The time elapsed from the start of the march to its stop is designated as the cessation time.

Claudication distance

This test is carried out like the previous one and differs from it in that the criterion taken as a measure is the distance traveled without claudication occurring.

Kisc test

With the patient lying down, he is asked to flex the thigh on the abdomen and the leg on the thigh at the same time, then proceeding to extend the limb. This exercise is repeated 30 times.

per minute. If it is positive, a painful cramp or discomfort will appear in the limb. Claudication will manifest more quickly in those patients with occlusive vascular disease.

STUDY OF THE CAPILLARIES AND PE UE AS ARTERIES

To carry out the study of the capillaries and small arteries, we can use several tests, among which we have:

- Plantar ischemia test.
- Paleness-blush test (Pratt).
- Angle of circulatory sufficiency.
- Reactive hyperemia.
- Capillary microscopy.

Plantar isemia test

Buerger highlighted the importance of the angle of circulatory sufficiency by studying the circulatory changes of the limbs at various angles of elevation.

Cehler reported some observations of color changes with both limbs at rest and after muscular activity.

Parke-Weber observed pallor of the feet in patients with circulatory disorders after repeated extension and flexion exercises of the foot. Regarding the pallor caused by these movements, Samuels developed what he described as proof of plantar ischemia. The test is performed as follows: the patient is asked to lie down and elevate both feet at a 90° angle.

You are ordered to perform rapid flexion and extension movements of your feet at a rate of 40 to 60 times per minute. You are instructed to flex your fingers at the same time. Good light is necessary for inspection of color changes in the plantar region and on the toes. This procedure is carried out for one minute. If the normal pink color of the feet persists, the result is said to be negative. If occlusive disease is present, a marked paleness will appear in the toes and plantar regions of the feet, then the test result is said to be positive.

This test is particularly notable when the involvement is limited to a single limb.

Pratt blush pallor test

Also called the postural color change test. These color changes are characteristic and can be produced in limbs with arterial insufficiency. With the patient lying down, elevating the leg produces very evident paleness, especially on the plantar surface of the fingers and the soles of the feet. When the patient sits with his feet dangling, the pallor is more or less replaced by a reddish purpuric erythema that may be diffuse or mottled.

In normal people with intact arterial circulation, a slight pallor may occur during elevation;
Later it gives rise to a normal color, around 30 s of the decline position. In a patient with arterial insufficiency, the return to normal color in the declining position will be considerably delayed (1 min or more), or the pallor is immediately replaced by flushing, as described above.

circulatory sufficiency angle

Buerger's angle of circulatory sufficiency is determined with the patient lying down, who is instructed to raise the leg slowly until it is placed at a right angle to the bed: in normal cases it retains its usual pink color. In patients who have a disorder of arterial circulation, the sole of the foot pales when elevated. When the patient slowly lowers the foot, the normal pink color returns. The angle that the leg forms in relation to the bed when normal color has returned is called the angle of circulatory sufficiency.

reactive iperemia

The extremity to be examined is elevated until its vessels are empty of their blood content (collapsed), as evidenced by the maximum pallor produced on the palmar or plantar surface. The cuff of a sphygmomanometer or a tourniquet is placed high on the extremity and a pressure above the systolic pressure is applied with the objective of occluding the arterial circulation; Then, the limb is placed in a horizontal position, 3 minutes later we remove the pressure exerted. The time necessary for a reddish color to appear (reactive hyperemia) and the time it takes for the skin to return to its normal color are noted.

In normal cases, below the cuff once the pressure is removed, a reddish rash immediately appears that progresses rapidly and uniformly to the distal portions, and reaches the fingers in a time of 10 to 15 seconds. The rash remains for 10 to 40 seconds and begins to disappear in the same order in which it appeared.

In organic or spastic arterial insufficiencies, the rash delays its appearance, progresses slowly, has a mottled appearance and may take 2 or 3 minutes to reach the fingers. Instead of being reddish, it may be cyanotic or take a long time to disappear.

Capillary microscopy

A drop of cedar oil placed on the skin makes it homogeneous and transparent, to the point that it allows visualization of the capillaries and blood flow.
I guide through them using a special microscope and lighting. The capillary system can thus be visualized at any point of the integuments including the mucous membranes.

STUDY OF THE GREAT ARTERIES

Among the tests we have for the study of the large arteries are:

- Oscillometry and oscillography.
- Venous filling time.
- Allen test.
- Measurement of systolic pressure in the extremities.

Oscillometry and oscillography

This examination method is based on the principle that if an optimal pressure, often between 80 and 150 mm, is applied through a cuff placed around the limb, the pulsations of the arteries passing at that level are transmitted to the air contained in the cuff; By connecting this to a device with a recording needle, the pulses can be visualized as oscillations of the needle against a dial or recorded on a plotting paper.

The amplitude of the observed oscillations has been taken as an index of the capacity of the large arteries that pass through there. On the contrary, decreased heart rate or readings of zero have been interpreted as indicative of decreased or insufficient circulation, suggestive of the presence of spastic or occlusive arterial disease.

The technique is the following:

1. The patient should be lying down and completely relaxed, with extremities exposed. Readings will be taken at several levels: thigh, leg and foot. Both limbs will be examined.

2. Air is pumped into the cuff above 120-140 mm recorded on the manometer.
3. When you press the key that activates the oscillometric mechanism, the needle will oscillate in a circular manner, back and forth in correspondence with the pulse.
   The reading will be taken at various levels, manipulating the valve and allowing the pressure inside the instrument to drop 20 mm each time the needle does not register the pulsation. For ease of reading we consider that the optimal pressure is between 120 and 80 mm. In hypertensive patients it can be started at 140 or 160 mm. The maximum oscillations are observed and considered as the oscillometric indices. The reading may vary according to the site or level of the limb.

Normal oscillomeric indices

iThigh, 4-16
iem roi erior Leg, 3-10
Foot, -4
iAxillary, 4-20; humeral, 2-12
iem ro superior Antebrachial, 1-10
Palmar, -2

Venous filling time

It was studied by Collens and Willensky. The patient is placed in a lying position; The limbs are raised to empty the veins on the back of the foot of blood, which is accelerated by flexion of the ankle and toes. The limb is then brought to the reclining position and observations are made of the time required to fill the veins on the dorsum of the foot. Normally this usually happens in 6 or 10 s. When the venous refill time is prolonged, it indicates that there is an occlusive vascular disease.

Allen test

The principles of plantar ischemia can also be applied to the upper extremities. Allen suggested compression of the radial pulse while the patient performs finger flexion and extension exercises for 10-15 s. If the ulnar artery is compromised, a definite ischemia appears taking the fingers and hand. A positive answer is very helpful, as it provides information about the ulnar artery, a vessel that is otherwise not easy to investigate. Allen suggested that this study should be included among routine examinations. It is common to find lesions of this vessel in cases of thromboangiitis obliterans, even in the absence of other symptoms.

Measurement of systolic pressure in the extremities

The measurement of systolic pressure in the extremities is a test that provides data of great importance, since it allows obtaining a general measure of vascular resistance above the measurement site.

Normally, a pressure gradient should be found in the extremities, which rises slightly from the femoral artery to the arteries of the foot, due to increased vascular sclerosis.

When any type of flow obstruction occurs, the blood must lose part of its energy in overcoming this obstacle and, therefore, it will reach the distal points with pressure values lower than in normal cases.

Different methods have been used to obtain pressure values in the extremities, among which the use of ultrasound stands out for its precision and accuracy.

STUDY OF VASOMOTOR TONE

To study vasomotor tone, the skin thermometry test is performed after anesthetic blockade of the paravertebral sympathetic ganglia.

Skin thermometry test

Paravertebral sympathetic ganglia block is commonly used to evaluate the sympathetic innervation of the upper and lower extremities. For this purpose, the entire territory from the first thoracic paravertebral sympathetic ganglion to the third is anesthetized with procaine. The appearance of a Claude-Bernard-Horner syndrome (enophthalmia, upper eyelid ptosis, effacement of the eyelid fissure, perikeratic conjunctival injection, miosis and anhidrosis of one side of the face), redness and dryness of the hand and increased skin temperature of the fingers indicate a positive test result.

In the lower extremities, the entire territory is anesthetized, from the first lumbar paravertebral sympathetic ganglion to the third or fourth. Signs of an effective blockage are: redness, dryness and increased skin temperature of the feet and particularly the toes.

There are three notable aspects of this procedure:

1. Elevation of skin temperature (usually 3-10 °C).
2. The increase in oral temperature (which is considered blood temperature).
3. The difference between these two values (which is attributed to the vasodilation of the small peripheral vessels).

For this increase in skin temperature to take place (vasodilation) , it is necessary that the sympathetic pathways be intact.

RADIOGRPHICAL EXPLORATIONS OF THE VASCULAR

Among the most important complementary exams for explorations of the vascular tree, we have:

Simple plate of blood vessels.
- Angiography.

Simple plate of blood vessels

Simple plaque of blood vessels can reveal calcifications of the vascular walls.
This procedure alone is not capable of informing about the permeability or otherwise of these vessels or the compensatory collateral

Angiography

The value of angiograms is incomparably higher than that of simple plaque of blood vessels. In fact, they will be able to accurately report the site of the lesion in the case of an occlusive or non-occlusive arterial disease. Angiography, moreover, makes it possible to obtain an exact notion of the degree of development of the compensatory collateral circulation. For the success of this exploration, the injection of a radiopaque substance into the lumen of the vessel to be studied is required:

According to the vessel being studied, angiograms can be divided into:

1. Aortography, if the aorta is the injected vessel.
2. Arteriography, commonly called opacification of the arterial tree of an extremity.
3. Phlebography, if visualization of the superficial or deep venous tree is performed.
4. Lymphography, if injected into the lymphatic system.

In the case of occlusive arterial diseases, arteriography allows us to locate not only the exact site of the occlusion, but also the location of its termination, so it indirectly informs about the extent of the lesion. Furthermore, it allows obtaining specific data on the degree of development of the collateral circulation in the case of non-occlusive arterial diseases, such as, for example, in arterial aneurysms and arteriovenous fistulas, where it can reveal not only the site of the lesion, but also its anatomical characteristics. This information acquires great value when planning the surgical strategy in these particular types of cases.

Phlebography can be performed by injection of the opaque medium into the venous system itself (direct phlebography). In other cases, the injection of the opaque medium is carried out via the arterial route (indirect phlebography), obtaining the exposures in the return phase of the opaque medium. Indirect phlebography can also be performed by injecting the opaque medium into the spongy substance of the bone (intrabone marrow phlebography). The injection of the opaque medium into the venous system can be carried out in the same direction as the venous flow (anterograde phlebography) or by injecting the opaque medium in the opposite direction to the venous flow (retrograde phlebography). If muscular exercise is performed during the examination, to demonstrate the venous pumping function assigned to it, it will then be known as dynamic phlebography.

Phlebography, in general, has great value in the diagnosis of the existence or not of old phlebitis in the deep venous sector, and also in the diagnosis of the location of insufficient communicating veins in those cases in which this diagnosis cannot be made clinically. Likewise, phlebography allows - especially when retrograde and dynamic techniques are used - to study the functioning of the valve system in the deep venous sector.

STUDY OF THE SUPERFICIAL VENOUS SYSTEM
AND THE SYSTEM OF THE COMMUNICATING VEINS
WITH THE DEEP VENOUS SYSTEM

Before going on to explain the tests to determine circulatory function in these systems, it is necessary to review the anatomy and physiology of the venous system of the lower limbs.

The venous system of the lower limbs is anatomically integrated by:

Deep venous system. It accompanies the homologous arteries, is located deeply (intermuscular) and is therefore intraaponeurotic. It conducts about 90% of the return blood from a limb.

Superficial venous system. Very variable in terms of its anatomical disposition. However, within this system two important veins can be prioritized: the internal and external saphenous veins, which flow into the femoral and popliteal veins, respectively. The superficial venous system is found in the subcutaneous cellular tissue, which offers little support. From this follows the fact that it is the usual seat of varicose vein syndrome0.

Under normal conditions, 10% of the blood return from an extremity is carried out through it.

Communicating (perforating) vein system. As its name indicates, this system puts the superficial and deep systems in communication. The number of communicating veins is very variable. The most constant are located in the upper third of the leg and in the lower third of the thigh. Under normal conditions they allow blood to pass from the superficial to the deep venous system and never in the opposite direction (fig. 48.1l).

The lumen of the venous system is located partly in stretch interrupted by conjunctivoendothelial folds, known as venous valves, which are constant at the level of the saphenous arch, as well as at the level of the communicating veins. The valve system from a physiological point of view obeys the law of the tension gradient, by virtue of which the valves will open, whenever the pressures predominate on the side of the valve that faces away from the heart chambers and, on the contrary, they will close, when the pressures predominate on the face of the one that faces the heart chambers.

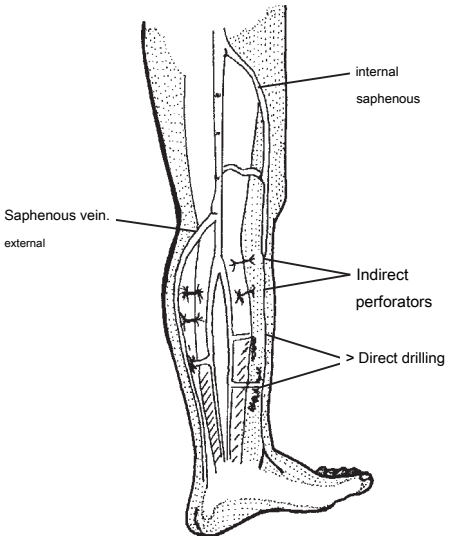


Fig. 48.1 Venous system of the leg.

Several factors intervene to guarantee centripetal progression - that is, against gravity - of the venous blood column. They are: negative intrathoracic pressure; the vision and fundamentally the valve system and muscle contractions. In effect, during the muscular contraction phase the deep venous system (which runs intermuscularly) is compressed with a consequent increase in intravenous pressure in that segment. If we remember the law of the tension gradient we will see that the blood will not be able to pass to the superficial venous system through the communicating veins or retreat into the deep venous system because the valve system prevents it (the pressure has increased on the valve side that faces the heart chambers and the valves close). Instead, the valve located proximally within the deep venous system will open (pressure has increased on the opposite valve face).

During the muscle relaxation phase, this venous segment, which has been left virtually empty, reduces its tension almost to zero and becomes an area of hypotension and aspiration, and obeying the law of the tension gradient, the valves will allow the passage of blood from the superficial system through the arches or communicating veins, as well as from the deep venous system itself in the distal portion of the segment we are considering. The valve system itself will prevent the blood that had already advanced into the deep venous system during the muscle contraction phase from returning to this segment.

If the ostial valve of the saphenous arch or of some of the communicating veins were insufficient, it would allow blood to pass from the deep to the superficial venous system (retrograde flow) during the muscular contraction phase. The superficial venous system would be seen forced to hold a greater amount of blood than its capacity allows, ending up dilating first and then elongating. In this way, varicose syndrome occurs due to valvular insufficiency. But there is also another type of varicose vein: sometimes venous dilation is due to a weakness of the venous wall itself, generally of endocrine cause, without valvular insufficiency. Varicose veins of slow evolution and accelerated development may appear, which are called arteriolyzed varicose veins and are due to the opening of arteriovenous communications. Both types require different treatments. Hence the importance of its differentiation.

Once the existence of varicose dilations is admitted, it is necessary to proceed with a careful study of them, with the aim of determining whether they are secondary varicose veins or primary or essential varicose veins, and in the latter case whether they are due to wall weakness or valve insufficiency. If it is a valve insufficiency, it is important to know if it corresponds to an insufficiency of the ostial valve of the saphenous arch or to a valve insufficiency in the communicating vein system. In case of insufficiency of the saphenous arch, it will be interesting to know which of them (the internal, the external or both) participates in the process. If there is valvular insufficiency at the level of the communicating system, the exact location of said communicating veins is of great importance.

There is a series of special tests aimed at clarifying these issues, but before pointing them out it is necessary to have knowledge of the following details:

1. When the limb is in a horizontal position, the communicating veins stop functioning in the normal individual.

2. When we elevate the limb above the horizontal, the superficial venous system empties of blood, leaving it virtually bloodless. This is of great importance not only in the clinical study, but also from a therapeutic point of view.

3. Once the superficial venous system is drained by elevation of the limb, it will take 30 s or more to refill when the upright position is assumed again.

4. Placing a tourniquet or ligature, or manual compression at a site on an extremity, suppresses circulation to the superficial venous system at that level. For the same reason, placing an elastic bandage will suppress superficial circulation to the entire bandaged area.

5. When an increase in intra-abdominal pressure occurs, a centrifugal current is generated by the compression of the cava and iliac veins, which lack valves.

venous vulae. This centrifugal pressure, however, is not transmitted to the lower limbs because it is prevented by the valve system, which functions as a shock absorber for such hypertension. The first valve structure is found in the external iliac vein, but it is inconstant.

Test to determine if these are primary varicose veins due to weakness of the venous wall or due to valvular insufficiency, Rivlin test

With the patient lying down, the limb is elevated above the horizontal to empty the superficial venous system of blood. The patient is then invited to stand up and the time it takes for the veins to fill again is noted. If they take 30 seconds or more, these are varices due to weakness of the venous wall, susceptible to being subjected to sclerosing treatment. If they are filled in less than 30 seconds, they will be varicose veins due to valvular insufficiency with retrograde flow of blood through communicating veins or insufficient arches that require surgical treatment.

Test for exploration of the internal saphenous arch Adams test

It consists of placing the pad of the index and middle fingers 2 cm below the femoral arch and 1 cm inside the beat of the femoral artery. The patient is made to cough to cause an increase in intra-abdominal pressure. If there is insufficiency of the ostial valve, a thrill will be perceived when the venous blood flow passes from the femoral valve to the internal saphenous valve.

Test to determine if it is an insufficiency of the internal saphenous arch or the communicating veins Trendelenburg test

With the patient in a supine position, the affected limb is elevated until the veins are empty. This can be completed with manual expression.
Once bloodless, a ligature is placed on the root of the thigh and the patient is invited to stand up. Several results can be obtained:

- Trendelenburg null.
- Positive Trendelenburg.
-Negative Trendelenburg.
-Double Trendelenburg.

Trendelenburg null. When the compression is removed, no change is observed and the varicose veins fill from the foot to the thigh in a time of 30 to 60 s (fig. 48.2). This result is common in incipient varicose veins without valvular insufficiency. It is equivalent to a negative Rivlin test.

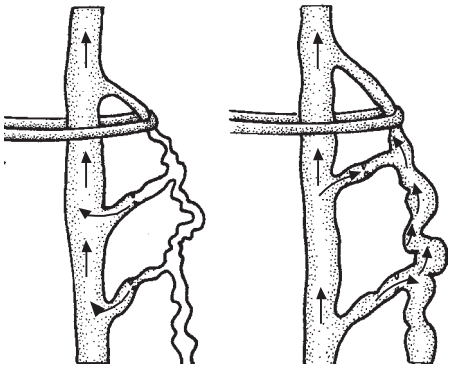


Fig. 48.2 Trendelenburg test: a, leg elevated; b, leg in standing position. The result is null.

Positive Trendelenburg. When the compression is removed, the collapsed veins suddenly fill from top to bottom. The staff is insufficient (fig. 48.3).

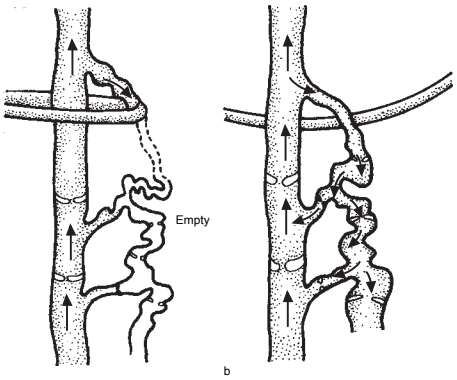
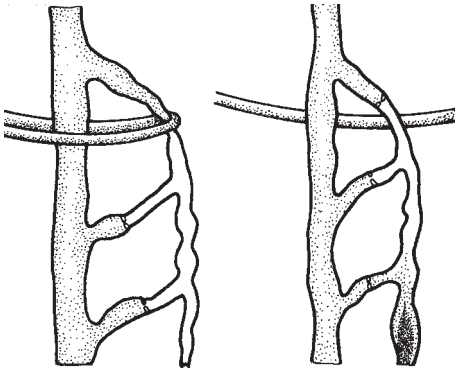


Fig. 48.3 Positive Trendelenburg test: a, leg elevated with compression; b, standing while removing compression.

Negative Trendelenburg. Collapsed veins fill before removing the compression; removing it does not change the venous filling. The staff is enough. The communicating veins are insufficient (Fig. 48. 4).

Fig. 48.4 Negative Trendelenburg test: a, standing, still with compression; b, standing while removing compression.



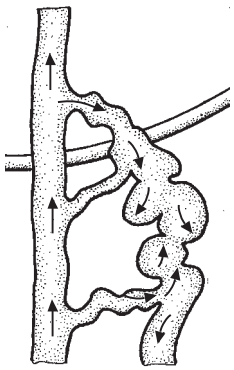


Fig. 48.5 Double Trendelenburg test.

Double Trendelenburg. Collapsed veins fill before compression is removed; When you remove it, the filling is even greater. In this case the communicating veins and the arch are insufficient (fig. 48.5).

Interpretation

1. By placing the ligature at the root of the thigh, circulation in the internal saphenous vein is suppressed, both in the centripetal direction, which is normal, and in the distal direction, which is abnormal.
2. Due to the arrangement of the valves in the communicating veins between the superficial and deep systems, normal circulation is carried out from the superficial to the deep system, and not the other way around. This allows us to understand that if when the person with the ligature at the root of the thigh stands up, it takes more than 30 seconds for the venules to fill, this is because the communicating veins are responsible and, therefore, they are undamaged.
3. When the system of communicating vessels is insufficient, the varicose veins can only be filled through the deep system and in these cases the test is null, since when the ligature is released the result is not reliable.
4. If we perform the test (ligation in the groin, patient standing) and the veins do not fill, we remove the ligation.
   If it fills rapidly from top to bottom, the test is positive, demonstrating that the internal saphenous valves are insufficient. Otherwise, it is called negative, since sufficient valves do not allow the retrograde passage of blood.
5. The combination of a null test and a positive test demonstrates double pathology, that is, damage to the valves and communicating veins.

Tests to determine the location of insufficient communicating veins

These tests are:

Fractional Trendelenburg test.

- Oschner and Mahhorner test.

- Pratt test.

Fractional Trendelenburg test. After the veins have been emptied, two or three tourniquets or ligatures are applied to different levels of the limb. The patient is stood up and it is observed which venous segments are filled. Insufficient communicating veins will be located between two of these ligatures, if the collapsed veins become suddenly engorged when the patient stands up.

Oschner and Mahhorner test. After venous emptying, an elastic band is placed on the root of the limb and the patient is ordered to walk. Result:

1. If the communicating veins are sufficient and the deep venous system is patent, the varicose veins will remain collapsed.
2. If the communicating veins are insufficient, the varicose veins will become engorged with the effort of walking.
3. The insufficient communicating vein will be located by descending the point of application of the ligature. There will come a point below which the vein will remain collapsed, since the insufficient communicating has remained on a higher plane.

Pratt test. Venous emptying is done by elevating the extremity and applying an elastic bandage to the middle part of the thigh; elastic compression is also performed on the root of the thigh (fig. 48.6). With the patient standing, the elastic bandage is removed from top to bottom, preserving the ligature across the thigh (Fig. 48.7). The location of the insufficient communicating vein will be known by the sudden filling of some varicose bundle when removing one of the turns of the elastic bandage (fig. 48.8).

Test to determine if the valvular insufficiency corresponds to the internal saphenous or the external
Ayerdale Anderson test

With the leg elevated, a rubber tourniquet is placed high around the thigh, or the examiner's fingers are applied at the level of the arch of the internal saphenous artery at the site of its opening into the femoral artery; At the same time, the external saphenous vein is occluded by digital pressure at the point where it empties into the popliteal vein; The patient is then invited to stand up with the aforementioned pressures maintained.

If varicose veins are the result of an insufficiency of the communicating veins; These will fill despite the occlusions applied to the internal and external saphenous veins.

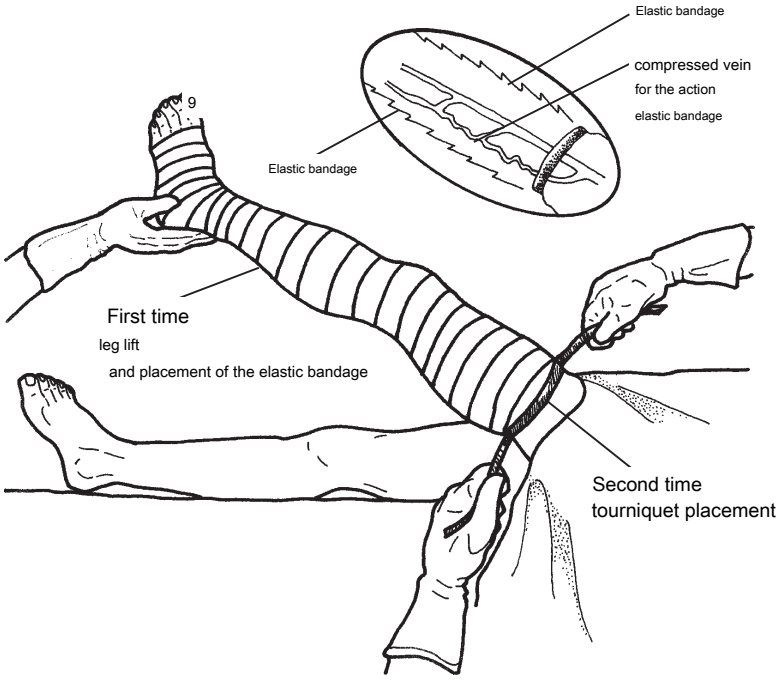


Fig. 48.6 Pratt test: first position.

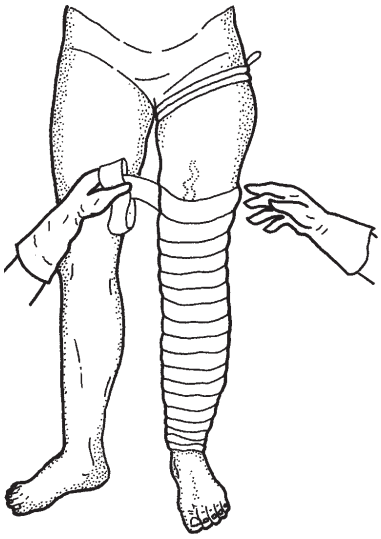


Fig. 48.7 Pratt test: second position.

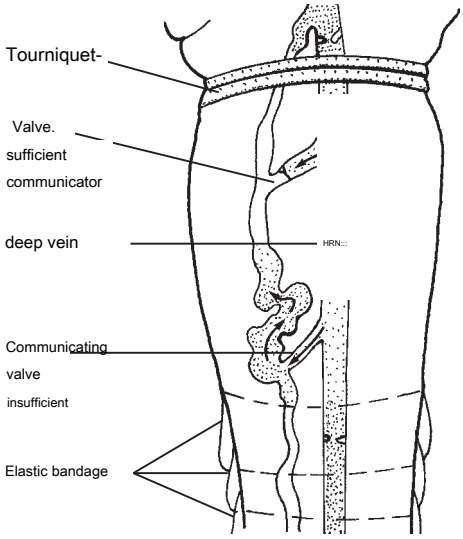


Fig. 48.8 Pathophysiology of the Pratt test.

If the varicose veins remain empty, it means that the defect is in one or both of the superficial veins. The tourniquet placed at the level of the thigh (or the pressure applied, if this was done with the fingers) is removed; If the varicose veins still remain empty, the internal saphenous vein is sufficient. If they fill, the internal saphenous vein is the source of retrograde blood flow. If the varicose veins remain empty after removing the tourniquet, but fill immediately after removing the pressure applied to the external saphenous vein, only this last vessel is insufficient.

STUDY OF THE DEEP VENOUS SYSTEM

In a case of secondary or post-phlebitic varicose veins, it is necessary to know whether or not there is permeability in the deep venous system, before proceeding to its removal.

Two tests can inform about the patency or not of the deep venous system. They are:

- Perthes test.
- Oschner deep permeability test.

Pert test is

While the patient is standing, an elastic compression is placed on the thigh (so as to interrupt the superficial venous circulation and not the deep one) and he is ordered to walk. If the varicose veins are depressed, it can be concluded that:

1. The deep venous system is patent.
2. The communicating veins are sufficient.
3. Varicose dilations of the superficial venous system do not fulfill a useful function, therefore, their resection or ligation will be beneficial for the venous circulation of the limb.

Oscner Deep Permeability Test

The veins are emptied by elevation, an elastic bandage is placed from the foot to the thigh, and the patient is ordered to walk for 15 or 30 minutes. Result:

1. If pain and cramps appear in the leg, there is obstruction of the deep venous system. The superficial veins cannot be resected, since they fulfill a supplementary function.

2. If the patient does not experience discomfort or, on the contrary, is relieved, the deep venous system will be patent and the superficial varicose veins should be treated.

Procedures for determining thrombosis of the deep veins of the legs

Due to the severity of its possible complication with pulmonary embolism, all efforts must be made to determine early thrombosis of the deep veins of the leg. Bedridden patients, who are potential candidates for intravascular occlusion, should be frequently and carefully examined; For this purpose, several tests must be done:

- Homans sign.
- Neuhoff sign.
- Rosenthal sign.

Homans sign. It is the most used of the procedures.

It consists of lifting the patient's leg with one hand and performing forced dorsiflexion of the foot. If the deep veins of the posterior compartment of the leg are thrombosed, the neighboring structures will press on the immediate nerves, producing localized pain.

Neuhoff sign. Keeping the patient lying on the bed, resting on his heels, knees bent.

If the muscles of the leg are tight and relaxed, the verification of a filling, infiltration or sensitivity at the level of the calves is considered a positive sign of venous thrombosis. This finding may exist in venous thrombosis even when the Homans sign is negative.

Rosenthal sign. This test has value in determining deep vein thrombosis, in the anterolateral compartment of the leg. It is performed in the following way: passive extension of the foot at 45° or less, and if this does not cause pain, then plantar flexion of the toes is performed. If the sign is positive, one should think.

sar in a thrombosis of the anterior tibial vein.

OTHER TESTS

In the study of the circulation of the peripheral vascular system, in addition to the tests already studied we have others.

circulatory speed

Circulatory velocity was originally studied in cardiac patients. It is also used, however, in peripheral vascular disorders. Calcium gluconate, saccharin, magnesium sulfate, ether, paraldehyde and, later, other substances such as fluorescein have been used for this purpose. The usefulness of circulation time as an aid in the diagnosis of peripheral vascular disorders is very doubtful, however, this test has great value in patients in whom an angiocardiographic study is to be performed.

Radioactive isotopes

Another method developed for the study of peripheral circulation has been the use of radioactive isotopes in arterial and venous circulation.

To evaluate the method, two techniques can be used, in general:

1. Obtaining images.
2. Obtaining isotope elimination curves.

For this purpose, different substances are used such as: I25, I5, streptokinase, labeled fibrinogen, technesium, etc.

It is important to note that the radioactive isotope method requires a considerable investment, since, in addition to obtaining data from the patient, it is necessary to protect the technicians who manipulate this equipment.

MAIN SYNDROMES OF THE VASCULAR SYSTEM

PERIPHERAL

49

The main syndromes of the peripheral vascular system are:

- Peripheral venous syndromes.
- Peripheral arterial insufficiency syndromes.
- Arterial aneurysms.
- Arteriovenous fistulas.
- Diabetic foot.
- Lymphatic syndrome.
- Raynaud's phenomenon. Raynaud's disease. Raynaud's syndrome.
-Acrocyanosis.
- Livedo reticularis.
- Erythromelalgia or erythermalgia.

PERIPHERAL VENOUS SYNDROMES

We will study the syndromes:

- Varicose veins.
-Venous obstructions: thrombophlebitis and phlebothrombosis.

V RICES

Concept

They are tortuous and diffuse dilation of the veins due to valve insufficiency.

Syndromog nesis or pathophysiology

The venous system of the lower limbs is anatomically integrated by:

1. Deep venous system.
2. Superficial venous system.
3. Communicating vein system.

If the ostial valve of the saphenous arch or of some of the communicating veins were insufficient, it would allow the passage of blood from the deep to the superficial venous system (retrograde flow) during the phase

of muscle contraction. The superficial venous system would be forced to hold a greater amount of blood than its capacity allows, ending up dilating first and then elongating. In this way, varicose syndrome occurs due to valvular insufficiency.

There is also another type of essential varicose veins in which venous dilation is due to a weakness of the venous wall itself, or to the congenital absence of numerous valves. Both types require different treatment, hence the importance of their differentiation, which is beyond the limits of this book.

Syndromography or positive diagnosis

Clinical picture

Varicose veins can be: superficial or deep. Superficial ones are easy to discover, as they appear as large sacciform dilations and obvious tortuosity of the veins. Some patients endure this without discomfort; others, however, may have pain.

If the varicose veins are deep, pain or fatigue in the calf muscles predominates, which is relieved by exercise and exacerbated by standing or sitting (the opposite of what happens when there is an arterial obstruction).

Chronic edema is another common symptom: it is firm and indurated.

If therapeutic measures are not taken, erythema, pigmentation, ulcerative lesions, etc. may occur.

complementary exams

The clinical tests used to demonstrate venous insufficiency and the most useful complementary tests have already been studied in the previous chapter.

Etiology

1. They are frequently due to a sustained increase in abdominal pressure (pregnancy, tumors, constipation).

2. Sequelae of phlebitis or vein thrombosis.

3. Congenital absence of numerous valves or weakness of the wall, apparently of a hereditary nature, as it is frequently observed in members of the same family.

VENOUS OBSTRUCTIONS THROMBOPHLEBITIS AND PHLEBOTHROMBOSIS

Concept and pathophysiology

The terms thrombophlebitis and phlebothrombosis are used to designate venous thromboses resulting from inflammation of the venous wall (phlebitis) and those derived from alterations in blood coagulation with or without circulatory delay, and hemaic alterations without or with parietal alteration.

Syndromography or positive diagnosis

Clinical picture

It is characterized by tachycardia, pain, functional impotence, edema, cyanosis (discreet) and manifest superficial collateral circulation. Once the disease is over, the following consequences remain: heat, edema, varicose veins, hyperhidrosis, erythrocyanosis and post-phlebitic ulcers.

Although there is no clear delimitation between the two processes, sometimes they are due to different causes and present some differences in the clinical aspect, as represented in Table 49.1.

Table 4.1

	Fle elsem or i	Trom o le iti
Etiology	Stasis and alterations	Inflammation of the blood wall venous
Coagulation intravascular	Primary	Secondary to phlebitis
Thrombus structure	Red	White or mixed
Location more frequent	Leg or foot	Thigh or pelvis
Symptoms	Little or no	Appropriate (fever, pain, edema, etc.)
Pulmonary embolism	Frequent	Rare
Sequelae	Rarely	Common

Thrombotic states (phlebothrombosis and thrombophlebitis) are, in most cases, identical and have the same complications, which is why it is difficult to make a differential diagnosis between the two. That's why that most authors accept only the name thrombophlebitis, since in the case of phleb0thrombosis, the adherence of the clot to the wall of the vein also causes an inflammatory reaction.

Etiology

Among the precipitating factors responsible for venous thrombosis of the lower extremities are trauma, infections and chemical irritation, but in a large group of patients we cannot find a defined cause.

Risk factors for venous thrombosis are:

serious illness, immobilization, bedridden, malignant neoplasms (especially in the pancreas, lung or digestive system), administration of certain drugs such as oral contraceptives, also appear in the postpartum, heart failure and postoperative period, among others.

PERIPHERAL ARTERIAL INSUFFICIENCY SYNDROMES

Peripheral arterial insufficiency can occur in two forms: chronic and acute.

CHRONIC PERIPHERAL ARTERIAL INSUFFICIENCY IS CHRONIC ARTERIAL INSUFFICIENCY

Concept and pathophysiology

It is the condition that results from those occlusive organic processes that present a chronic form of interruption of blood flow in an extremity. Circulatory impoverishment may be associated with a vasospastic disorder or organic changes in the blood vessel wall due to sclerosis or inflammation; As the obstruction installs slowly, it allows the development of collateral vessels, sometimes in large proportion, which masks the condition by presenting few symptoms, and that is the reason for using some tests to reveal it.

Other times the symptoms are triggered by cold or sudden exertion.

Ischemia can be unilateral or bilateral, depending on the site of the anatomical lesion.

Syndromography or positive diagnosis

Clinical picture

The most important symptoms and signs of the clinical syndrome are: fatigue, intermittent claudication, pain, paresthesia, color changes (paleness, redness, cyanosis), trophic changes in the skin (hair, nails or muscles), coldness, gangrene and decrease or absence of pulses and oscillometric indices.

complementary exams

The most valuable complementary exams are:

1. Arteriography (to specify the level of the obstruction).

2. Systolic pressure in the extremities.

3. Hemodynamic studies: pressure indices; segmental pressures; digital plethysmography.

Etiology

It is observed in:

1. Arteriosclerosis obliterans.

2. Thromboangitis obliterans.

ACUTE PERIPHERAL ARTERIAL INSUFFICIENCY ACUTE ARTERIAL UMEMIA

Concept and pathophysiology

It is the clinical picture that results from the complete and sudden obstruction of blood flow in an extremity. Its main causes are thrombosis and embolism. Acute spontaneous thrombosis of an artery commonly occurs as a complication of arteriosclerosis obliterans. Acute arterial embolism produces the same clinical picture and occurs in individuals who suffer from cardiovascular diseases, which give rise to the formation of centrally located thrombi (more frequently in the left chambers of the heart).

Syndromography or positive diagnosis

Clinical picture

It is characterized by: acute and lancinating pain, paresthesias, functional impotence, hypoesthesia or anesthesia, sudden coldness and color changes (pallor, cyanosis)

complementary exams

Absence of pulse and oscillometric indices below occlusion.

Etiology

Mitral stenosis, atrial fibrillation and other heart diseases are what cause the following: acute arterial thrombosis and acute arterial embolism.

ARTERIAL ANEURYSMS

CONCEPT

An aneurysm is the abnormal and permanent dilation of an artery that occurs as a result of weakness and widening of its walls. This definition excludes dissecting aneurysm and false aneurysm or pulsating hematoma. Aneurysms can appear during the course of arteriosclerosis or syphilis. They can also be traumatic or congenital.

SYNDROMOGRAPHY POSITIVE DIAGNOSIS

Clinical picture

The patient almost always consults due to the presence of a tumor with pulsation and expansion, and due to abdominal or lumbar discomfort that simulates renal colic, in the case of aortic aneurysms; Sometimes they arrive at the doctor in a state of shock due to the rupture of the tumor or with an acute abdominal condition. When the aneurysm is located in the upper or lower limbs, the symptoms are early: a tumor in the course of an artery, which expands synchronously with the pulse, almost always painful, with a decrease or absence of pulses below it.

A syndrome of chronic or acute ischemia may occur, or it may also debut due to a rupture or fissure.

On auscultation and palpation there may be a murmur and systolic thrill, respectively.

Complementary exams

Aortography and arteriography corroborate the diagnosis.
nostic.

ETIOLOGY

The aneurysm is almost always caused by arteriosclerosis, syphilis, trauma, etc.

ARTERIOVENOUS FISTULAS

CONCEPT

An arteriovenous fistula is understood to be the direct abnormal communication between an artery and a vein with the consequent return of blood to the cardiac chambers without passing through the capillary bed.

Fistulas can be congenital (multiple and small) or they can be acquired, almost invariably produced by penetrating wounds that lacerate the artery and vein, establishing communication; This can be direct or through an aneurysm sac.

SYNDROMOGRAPHY OR POSITIVE DIAGNOSIS

Clinical picture

It can remain ignored if a good physical examination is not performed; The symptoms are usually varied, sometimes vague, such as heaviness, dullness of the limb, or sometimes more or less persistent pain, varicose veins or unilateral edema and trophic disorders (ulcers and gangrene).

Auscultation allows you to hear a continuous murmur with systolic reinforcement or machinery noise. On constant palpation you will find a shudder.

net vibratory foundation, the thrill, which will always be continuous with systolic reinforcement.

Complementary exams

Determination of oxygen saturation in venous blood, arteriography and telecardiogram.

ETIOLOGY

Let us cite as examples multiple congenital arteriovenous fistulas and post-traumatic arteriovenous fistulas.

DIABTIC FOOT

CONCEPT

It is the injury that occurs in the foot of diabetic patients due to the association of macroangiopathy, neuropathy and microangiopathy.

SYNDROMOGENESIS PATHOPHYSIOLOGY

Diabetes mellitus is accompanied by multiple chronic complications where circulatory alterations in peripheral blood vessels have a great specific weight. Diabetic foot lesions are produced by a mosaic" of pathological combinations in the same patient.

Neuropathy. It is related to alterations in sensitivity, fundamentally the anesthetic or hyposthetic zone.
The involvement of neuropathy on vasomotion (change in caliber of blood vessels) has also been invoked as an etiological agent.

Microangiopathy. Atherosclerotic lesions in the arteries of the lower extremities manifest earlier in the diabetic as a consequence of chronic metabolic alterations: dyslipidemia, enzymatic glycosylation of lipoproteins, decrease in prostaglandins at the level of the endothelium and increase in thromboxane A2, progressive vascular calcification, as well as the secretion of oxidant and vasoconstrictor substances, all acting at the level of the arterial wall.

This lesion of the lower extremities, which is frequently observed in the kidney and retina of diabetics, is currently the subject of studies. Early in the life of the diabetic, microvascular flow translates into a thickening of the basement membrane, which promotes sclerosis and limits the ability to vasodilate the microvasculature, preventing hyperthermic responses and interfering with the self-regulatory response (vasomotion).

SYNDROMOGRAPHY OR POSITIVE DIAGNOSIS

Clinical picture

There are two fundamental types of diabetic foot:

1. Neuroinfectious:

a) Abscess.

b) Phlegmon diffuse.

c) Cellulite.

d) Bad diabetic plantar perforator.

The increase in volume, pain on palpation, local heat, fluctuation of the turgid areas where the secretion is deposited, with febrile symptoms or not, accompanied by a physical examination where the peripheral pulses are all palpable and the neurological examination shows hypoesthesia or persistent anesthesia, is the clinical picture that accompanies this type of diabetic foot.

The bad plantar perforator deserves a separate chapter, an ulcerated lesion that bleeds easily, has thick edges, is painless and where the sepsis factor is easily associated. This type of injury develops in the support points of the diabetic's foot.

2. Ischemic:

a) The ischemic ulcer.

b) Ischemic gangrene.

The ischemic ulcer appears preferentially on the balls of the toes in the interdigital areas.
Its main characteristics are: pain, atonic background of the ulcer, accompanied or not by infection.
Ischemic gangrene is manifested by the necrotic area of the distal portions of the toes; the lesion is progressive according to the progression of the disease. On clinical examination, one or more peripheral pulses were not detected, and the arterial tests described above were positive.

Specialized complementary exams

Hemodynamic studies, simple X-rays of the bones of the foot. Angiographic study. The other complementary ones according to their underlying disease: diabetes.

ETIOLOGY

External mechanisms (generally traumatic) caused in the foot of the diabetic, with involvement of the macro and microvasculature, and associated neuropathy and sepsis.

LYMPH TIC SYNDROME

We will study two types of entities in this syndrome:

- Lymphedema.

- Acute lymphangitis.

LYMPHEDEMA

Concept

All edema is the result of an imbalance between capillary filtration and lymphatic flow. When edema occurs as a result of a failure in lymphatic drainage, the disease is called lymphedema.

Syndromog nesis or pathophysiology

It may be due to an intrinsic abnormality of the lymph conduction pathways (primary lymphedema) for which no external cause can be found, while secondary lymphedema arises from factors originating outside the lymphatic system, for example:
surgery, radiotherapy and infection. Lymphatic drainage can fail due to:

1. Hypoplasia or aplasia of lymphatic vessels, of congenital or acquired origin.

2. Obstruction due to scarring or tissue fibrosis.

3. Poor contractility of lymphatic collectors.

4. Dilation of the lymphatic collectors causing loss of appetite.

Syndromography or positive diagnosis

Clinical picture

According to the degree of lymphedema, four clinical stages can be considered:

Stage I. Soft edema, without skin lesions. Remits With treatment in three months.

Stage II. Soft edema without skin lesions. Does not resolve with treatment in three months.

Stage II. Hard and fibrous edema (fibredema), there are no postural changes or presence of skin lesions.

Stage IVo elephantiasis. Presence of skin lesions and significant fibrosis. The presence of skin lesions (hyperkeratosis and papillomatosis) are essential to use the term elephantiasis. These lesions do not have to affect the entire limb and may appear localized in regions, especially at the level of the dorsum of the fingers and distal areas of the limb.

complementary exams

- Direct or radioisotopic lymphography.

- Xeroradiography.

- Computed axial tomography.

- Nuclear magnetic resonance.

- Two-dimensional ultrasound.

Etiology

It can be caused by a congenital problem or acquired as a result of a surgical or infectious process.

ACUTE LYMPHANGITIS

Concept

Acute inflammation of the lymphatics of the dermis due to pyogenic infection that invades them through an entrance door, generally affecting the lower limbs and can also be caused by a distant focus of infection.

Syndromography or positive diagnosis

Clinical picture

The patient begins to have discomfort in his extremity about three days before appearing a picture of general non-specific discomfort, weakness, nausea and vomiting, headaches, chills precede high temperatures that subside within 72 hours. The extremity presents an increase in volume, redness, heat, itching and in the inguinal region a turgid and painful lymphadenopathy develops, even without being palpated. This symptomatology will be more acute depending on the clinical form it corresponds to:

Reticular. The most frequent. Diffuse edema, heat and redness in the affected tract.

Flictenular. Blisters appear on the skin.

Truncular It manifests itself with very consistent, hard, reddish cords that reach the lymph nodes.

Necrotizing. The most serious clinical form. Flictenae appear filled with serosanguineous secretions. They break and whitish tissues appear that require surgical treatment.

Deep. Pain on pressure in the vascular tracts and adenitis. May cause thrombophlebitis.

Etiology

Pyogenic infection of the dermis due to streptococcus (generally) or fungi, parasites and viruses.

RAYNAUD PHENOMENON

CONCEPT

Raynaud's phenomenon is the active and intermittent occlusion of the small digital arteries that carry blood to the extremities. It is clinically manifested by discoloration or paleness and/or cyanosis of the affected parts, followed by red discoloration. It is accompanied by frequent numbness and a decrease in temperature in relation to the phase.

The three typical phases of the process are: pallor or local syncope, cyanosis, or local asphyxia, and flushing or reactive hyperthermia.

In 1861, Maurice Raynaud published a thesis entitled On local asphyxia and symmetrical gangrene of the extremities, in which he described paroxysmal changes in the color of the fingers, which could be further complicated by trophic disorders and even gangrene. He explained these disorders caused by cold and emotions by vasomotor phenomena of nervous origin without organic lesions of the arteries or veins. The autonomy and pathophysiology of this disease were quickly questioned, since numerous examples of the phenomenon described by Raynaud were observed during the course of various organic conditions, particularly of the skin and arteries.

Despite everything, Raynaud's disease of unknown etiology remains, that is, without a detectable organic cause, which corresponds to the author's pathophysiological conception, which is bilateral and of unknown cause, and Raynaud's syndrome, in which vasomotor disturbances are secondary or are associated with well-defined diseases. Symptoms are almost always unilateral.

Modernly, these three aspects are considered with the designation of functional arteriopathies due to vasoconstriction, along with acrocyanosis and livedo reticularis.

SYNDROMOGRAPHY OR POSITIVE DIAGNOSIS

Clinical picture

In its usual form, Raynaud's disease is manifested by paroxysmal attacks in which two phases are distinguished: syncopal and asphyxia. It is of interest to the fingers of the hand, more rarely the feet and, exceptionally, the tip of the chin, the nose and the earlobes. The syncopal phase is manifested by progressive paleness and coldness of the fingers, from the tip to the root, with predominance in the last two phalanges. Sometimes the pallor is greatest from the beginning (dead finger phenomenon). These alterations are symmetrical and are accompanied by numbness of the fingers, which become insensitive and clumsy; One or more fingers may be of interest, excluding the thumb.

The asphyxia phase follows the previous one. It is characterized by excessive overheating, itching and tingling, sometimes with burning and throbbing pain. At the same time, the pallor is replaced by cyanosis (purple red) that

Follow the same progression, from the end to the root of the fingers. Only some of the elements indicated may be presented; Its duration is variable.

These phases are triggered by cold, but the sensitivity threshold differs in different subjects. They can be reproduced experimentally by immersing the hands in cold water, therefore, the evolution is seasonal, getting worse in winter and improving in the warm season. In 25% of cases they are also caused by emotions (anger, surprise).

Raynaud's phenomenon is considered as common as high blood pressure or diabetes mellitus, and climatological influences seem important. In French Savoy, 16.8% of the general population is affected, while in South Carolina, an area with a mild climate, its prevalence is 5% and in Spain 3. 7%.

Complementary Exams

The most frequently performed tests are:

the anatomopathological study and the cold test.

SYNDROMOGENESIS OR PHYSIOPATHOLOGY

The paleness is due to spasm of the arterioles and partly of the venules. The capillaroscopic study carried out in this phase allows us to observe that blood does not enter the capillaries. In the second phase, cyanosis is produced by blood stasis at the level of the capillaries and venular dilation with reflux of blood to the capillaries.
In the second phase, the final redness would be explained by reactional hyperemia with blood entering the arterial capillaries and recovery of normality. The mechanism by which these alterations occur is discussed.

ETIOLOGY

Various circumstances can cause or trigger Raynaud's phenomenon:

1. Repeated microtrauma (professional or sports activity).
2. Arterial occlusive disease, such as arteriosclerosis obliterans, thromboangiitis, embolism, thrombosis.
3. Collagen diseases, mainly scleroderma.
4. Metal poisoning (arsenic, thallium, lead), medications (ergotamine, beta blockers, nasal sympathomimetics, bleomycin); tobacco and LSD.
5. Blood disorders such as cryoglobulins, cryoagglutinins and increased viscosity (polycythemia, thrombocythemia, leukemia).
6. Cervical rib compression syndromes, scalene syndrome, costoclavicular compression and carpal tunnel syndrome.
7. Diseases of the nervous system such as poliomyelitis, peripheral neuritis, syringomyelia, hemiplegia, monoplegia, spina bifida and tumors or prominences of the intervertebral discs.
8. Other causes, such as myxedema or primary pulmonary hypertension.

ACROCYANOSIS

CONCEPT

The term acrocyanosis was introduced by Crocq in 1896 to designate a clinical situation characterized by persistent coldness and cyanosis of the distal parts of the extremities in the absence of pain.

SYNDROMOGENESIS OR PHYSIOPATHOLOGY

The mechanisms that produce vascular changes are unknown. A decrease in the caliber of the arterioles and a reduction in blood velocity, as well as dilation of the capillaries and venules, are confirmed, but the cause of these alterations is unknown.

SYNDROMOGRAPHY OR POSITIVE DIAGNOSIS

Clinical picture

It is a permanent vascular syndrome, which preferentially affects women. Although it sometimes appears in childhood, it usually begins during puberty, generally before the age of 20, with bilateral symmetrical manifestations characterized by the presence of cold, purplish extremities. The condition predominates in the upper extremities, in the fingers and hands and to a lesser extent, in the toes. The cold does not act as a trigger but rather as an aggravating factor of the symptoms, which are permanent, although less accentuated in hot weather.

Normally there is no pain, although in winter, and with very cold temperatures, swelling may occur, with sensitive and painful areas. There is always hyperhidrosis in the palm areas, sometimes very intense and aggravated by emotional situations; They generally increase during the hot season. The disease is unpleasant and causes aesthetic problems, especially when there is intense hyperhidrosis. This can go to the extreme of forming a real drop of sweat that makes daily life difficult and represents a stress situation that aggravates the process.

ETIOLOGY

The etiological factors of acrocyanosis are unknown. Occasionally, cases associated with

an endocrine dysfunction. It has also been observed in patients with schizophrenia or mental deficiency, but it has not been proven that these processes are etiological factors.

LIVEDO RETICULARIS

CONCEPT

It is an acrosyndrome characterized by a bluish-red mottled discoloration in the form of a fine reticulum of the skin of the extremities. It has been described with the terms livedo racemosa, livedo annularis and asphyxia reticularis.

Currently there is the criterion of separating these terms into two groups:

1. Livedo racemosa, in which there are morphological alterations in the vascularization of the skin, which is why it is also known as racemosa vasculitis.
2. Livedo reticularis which would be an exclusively functional disorder without morphological substrate.

SYNDROMOGENESIS OR PHYSIOPATHOLOGY

The pathophysiological changes consist of a functional or organic narrowing of the arterioles with dilation of the capillaries and venules. Spastic or functional obstruction of the arterioles affects only the peripheral capillary arborizations, which leads to skin discoloration with capillary atony and decreased flow velocity.

PATHOLOGICAL ANATOMY

Anatomopathological studies have revealed the existence of intimal proliferation in isolated arterioles and small arteries. In some cases, complete obstruction has been found, not only of the arterioles but also of the venules. Lastly, hypertrophies of the muscular layer have been reported.

SYNDROMOGRAPHY OR POSITIVE DIAGNOSIS

Clinical picture

It usually manifests itself between 20 and 30 years of age due to the existence of a persistent reticular, bluish or bluish-red mottled discoloration of the skin of the legs and, sometimes, the lower part of the trunk. The upper extremities are affected to a lesser degree. It is accentuated during winter and after exposure to cold but does not disappear in hot weather. Some patients report coldness, numbness and paresthesia of the feet and legs, but these signs are not common. In certain cases, ulcers appear on the skin of the legs or feet in cold weather.

The differential diagnosis can include acrocyanosis, thromboangiitis obliterans, periarteritis nodosa and scleroderma.

Complementary exams

The results offered by the ultrasonic flow velocity detector (Doppler) and capillaroscopy are normal. The biopsy performed in a healthy area will reveal the existence or not of the aforementioned structural changes.

ETIOLOGY

Among the most important etiological factors are high blood pressure and emotional instability. Cases associated with arsenic and lead poisoning, embolization of dermal arteries by cholesterol crystals from atheromas located in the abdominal aorta, and in the course of treatment with amantadine for Parkinson's disease have been reported.

Livedo reticularis has also been related to the presence of criglobulins, systemic lupus erythematosus (SLE) and anticardiolipin antibodies, typical of primary antiphospholipid syndrome.

ERYTHROMELALGIA OR ERYTHERMALGIA

CONCEPT

It is a vascular disorder characterized by episodes of elevated skin temperature of the extremities, accompanied by redness and burning pain, which occur when the skin temperature rises to a critical level, generally between 31 and 32 °C.

Erythromelalgia or erythermalgia is a dystonic and paroxysmal vascular acrosyndrome, characterized by the triad of red color, heat and pain, appearing in the form of a crisis, which leaves no sequelae. The first case was described by Graves in 1834, and Mitchell in 1878 suggested the term erythromelalgia. Smith and Allen in 1938 coined the term erythermalgia to highlight the increase in temperature in the red and painful areas of the affected limb. Both terms have been used indiscriminately, although it is the latter that best expresses the clinical data of heat.

SYNDROMOGENESIS PATHOPHYSIOLOGY

The exact pathophysiological mechanism is unknown. Obviously there is an increase in peripheral blood supply. Increased circulation in the vessels produces redness and increased skin temperature. This new factor may be responsible for the burning pain.

An exaggerated reaction to color in small vessels (dilation and increased capillary pressure) has been reported.

SYNDROMOGRAPHY OR POSITIVE DIAGNOSIS

Clinical picture

The episodes are characterized by the appearance of burning pain with redness in the most distal parts of the limbs, it is commonly bilateral and preferably affects the fingers, heel and the plantar region at the level of the support areas.

The episodes are of short duration and appear preferably in summer; They are usually associated with hyperesthesia and the patient cannot tolerate even the friction of bedclothes. They are precipitated by heat, standing or sitting with the foot hanging, position-dependent increase in hydrostatic pressure, local friction, and exercise. They are relieved by elevation of the extremities and cold.

On physical examination we found increased skin temperature, sustained flushing, palpable peripheral pulses, normal or elevated oscillometric indices, and absence of trophic disorders.

Erythromelalgia preferentially affects women, in whom it appears, above all, in middle age. Idiopathic forms usually manifest in childhood or in young people and have a tendency to present bilateral location, while in secondary forms unilateral location dominates and the onset is usually after the age of forty. The disease

It evolves into crises, triggered by changes in temperature, that affect the hands or feet, although sometimes they are limited to a single segment of a finger.

Complementary exams

History is usually sufficient to establish the diagnosis. The examination detects red or cyanotic red color, with heat and a certain degree of edema. It is possible to reproduce the crisis by applying heat or placing a pneumatic compression cuff on the root of the limb, which is inflated until the systolic pressure values are exceeded.

ETIOLOGY

In principle, two forms are admitted: idiopathic and secondary. Idiopathic occurs in healthy people who do not present signs of disease in the nervous or vascular system and the secondary form appears in cases of polycythemia and in states of blood hyperviscosity.

It has also been observed in patients with venous insufficiency, diabetes, hyperthyroidism, arterial hypertension, pernicious anemia and astrocytoma, systemic lupus erythematosus (SLE) and rheumatoid arthritis.

In 1988, an epidemic outbreak associated with pharyngitis and poxvirus isolation was reported. Cases secondary to poisoning by heavy metals (mercury and arsenic) have been described and subsequently in patients treated with nicardipine, nifedipine, bromocriptine, as well as during pregnancy. Familial cases have been observed, but the inheritance is not known.

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CLINICAL PROPEDEUTICS AND MEDICAL SEMIOLOGY (two volumes) combines the detailed exposition of the techniques for performing a complete physical examination in a supposedly healthy adult, with the study of the symptoms, signs and syndromes of general, regional and systemic semiology, which may occur in a sick individual. Furthermore, it points out, without going into depth, the most important paraclinical examinations in verifying the semiological or etiological findings, or

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